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TRAUMA

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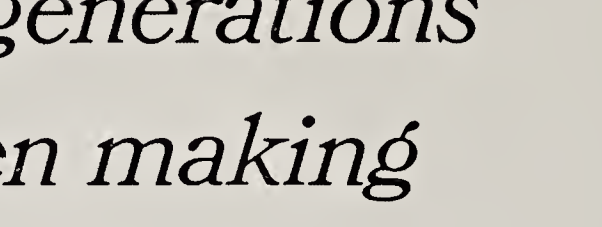

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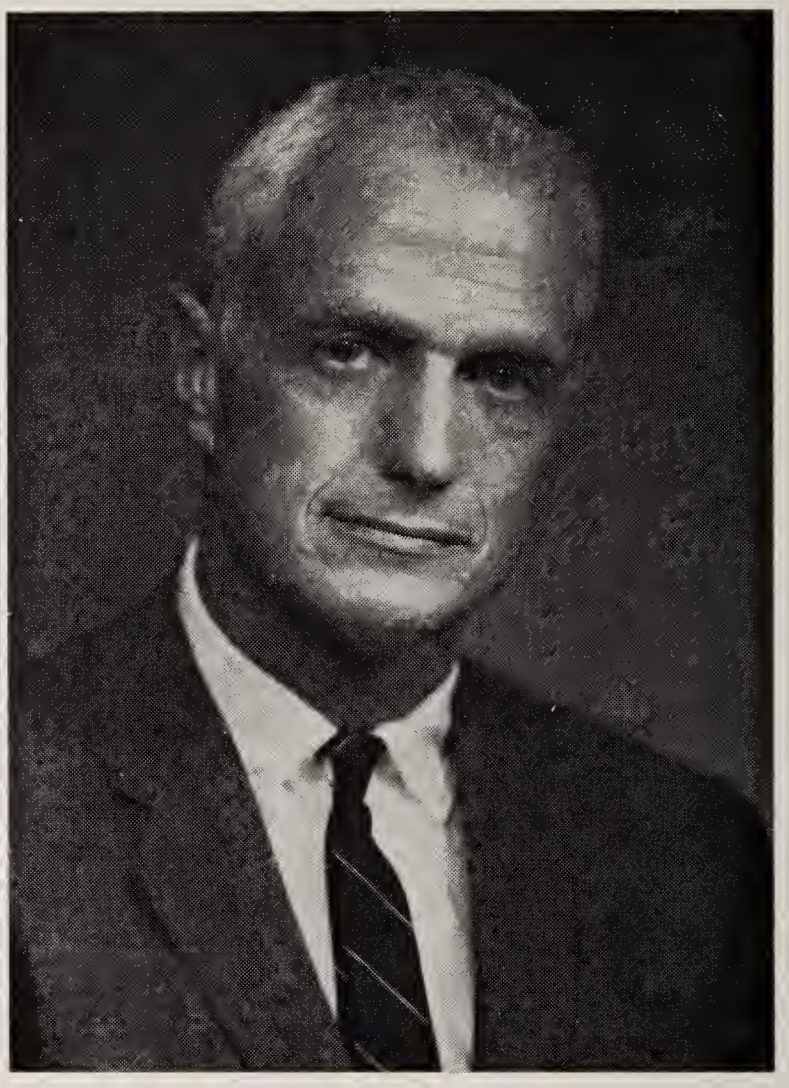
Born in Chicago, Illinois, Dr. Drucker obtained a B.S. at Harvard University and his M.D. at Johns Hopkins University School of Medicine in 1946. His post graduate work in Surgery was done at Johns Hopkins Hospital, the New Haven Hospital, Western Reserve University and for three years in the University Hospitals of Cleveland. He is a diplomate of the American Board of Surgery and a fellow of the American College of Surgeons. He became a professor of Surgery at Western Reserve University School of Medicine in 1966 where he had taught since 1954.

In 1966 he was appointed Professor and Director, Department of Surgery in the University of Toronto and Surgeon-in-Chief, Toronto General Hospital.

He is on the Board of Managers of the American Association for the Surgery of Trauma and on the editorial board of both the Canadian and American Journals of Surgery. Dr. Drucker is the author of several papers on a wide variety of topics. His current interests include haemorrhagic and endotoxic shock, especially the general metabolic and endocrine changes accompanying shock.



Guest Editorial

WILLIAM R. DRUCKER, M.D.
Professor and Director, Department of Surgery

I should like to die as a result of a gun shot wound inflicted by a jealous husband when I am over ninety. My chance of achieving this objective, like so many others, is slight indeed. But, if I must die soon, the odds are heavily in favour that it will be as the result of some form of trauma. These odds are even more heavily weighted for most readers of this journal. Your greatest chance of dying until you become a Professor, and thereby have questionable usefulness to society, is from some form of TRAUMA. A few may achieve the dubious distinction of a fatal ending in a crime of passion; most will die as the result of another form of passion that is not considered to be criminal.

To possess and drive an automobile has become an almost inalienable human right in Western Civilization. Territorial imperative, rather than sex, has become the working dogma for social scientists and for a few enlightened psychiatrists. Territory includes more than just real estate; for the folk hero of the western frontier it certainly included his horse and gun. For the cowboy of the asphalt jungle, for the pretty young miss with a bachelor apartment, for you, and indeed for me, a car is a very real part of our territory. It is essential for our practical existence, and perhaps for our emotional security.

It is interesting that with exactly the same blithe uncritical assurance with which society expects the recipient of an M.D. degree to be endowed with a capacity to teach, mere possession of a vehicle is somehome considered to confer upon the owner a capacity to drive it safely. However, insurance companies know better; ask any patient with a son younger than twenty-five years abou the financial advantage of driver training courses. The high insurance premiums required of youthful drivers do not reflect an avaricious private enterprise. Public indifference does not hide from agencies that ensure against loss from auto accidents the stark reality that this week, next week, and each week thereafter the direct measurable cost of highway and automobile death and injuries in this country will approximate 18 million dollars. In contrast, Toronto will have less than 10 million dollars to cover capital expenditures for all health facilities for the next fifty-two weeks!

GUEST EDITORIAL

Students of human behaviour say protective thinking, lawyers mention rationalization, politicians less kindly suggest an ostrich — all refer to the attitude "it can't happen to me". Perhaps, that is what Mr. H. H. Bliss thought as he stepped from a horse-drawn trolley seventy years ago in New York City to be run down by a horseless carriage and thereby claim his place in history as the first known human traffic fatality. If every person now living in Toronto could be considered a traffic fatality the total population of the metropolitan area would almost equal the 1,750,000 that have been killed by automobiles in North America. More than six years of war in Viet Nam has killed 35,000 Americans; 55,000 die annually on U.S. highways and more than 5,000 perish each year on Canadian roads. For every casualty in Viet Nam, six die from an accident in the U.S. For every person killed by an accident, ten to fifteen require hospitalization from a day to many months. Accident patients require 22 million bed days a year, or 12% of the total general hospital space. This amounts to more bed days than are required to take care of the deliveries of babies born each year, more than needed by all heart patients and four times more than used for cancer patients. According to former M. P. Howard Grafftey, the Conservative party's leading authority on highway and automobile safety, every second car rolling off the assembly line today will be involved in an accident resulting in death or a severe debilitating injury. One person in two hundred will require medical care for a road injury each year and as a result, on any given day, approximately one percent of the working population is disabled.

What can be done about this? The leading cause of death from shortly after birth until well into middle age is trauma. Almost half of trauma deaths result from the motor car. In the idealistic British Isles, the institution of a law requiring a breathalizer test to detect the presence of alcohol caused a significant abrupt reduction in traffic accident morbidity and mortality. As the practical realities of implementing the law became apparent to a population no less concerned with territorial imperative than their cousins on this side of the Atlantic, the morbidity figures began to rise again. The death rate from highway crashes has been reduced 80% by the combined use of lap and upper torso safety belts. This statistic gains validity when one recognizes that the overwhelming bulk of occupant injuries and death occur at crash speeds under 60 m.p.h. In Viet nam the mortality rate of soldiers injured by any of the ingenious devices employed in modern warfare is less than 1% among casualties arriving in hospital areas. No civilian population has approximated the speed of recovery or finesse of therapy achieved by the medical corps in the Armed Services.

Viewed in the concept of modern medical teaching, the patho-physiology of this epidemic is relatively simple. Very few cars kill or injure people in the garage; what happens in a garage is a matter of personal freedom. However, once on the highway a driver interacts with innumerable other individuals — each endowed with his own sense of territorial imperative. At this time a driver is no longer free. Car design and road engineering have provided more safety than ever before, but while improved highway engineering, more safety features in automotive design, compulsory periodic retesting of drivers and cars, and stricter laws are all necessary concerns, they are peripheral to the basic problem. Until society considers a driver of a car not licenced to kill, the leading cause of death from slightly after birth until well into middle age will continue to flourish.

As physicians we remain concerned with the therapy of accident victims. This is our necessary and proper role, but as in so many other medical disorders the question might properly be asked whether our time might be more profitable spent on preventive measures rather than on the therapy of specific individuals. In the long run this may gain us more free time to enjoy the pleasures of relaxing — in a car.

There will be five issues in this volume of the JOURNAL. Changes in the editorial format are minimal. Each issue contains some general review articles on a broad theme. Clinicopathological Conference, Recent Advances, Student Research and Symptomatology are retained as regular features.

Articles will tend to emphasize the synthesis of the basic medical sciences into clinical practice. In an attempt to broaden the scope of this publication as ageneral review JOURNAL, Grand Round Quiz has been introduced as a new feature. It is designed to reinforce some of the more salient aspects of the theme of each issue.

Titus Owolabi

Doctors, Accidents And Accident Prevention

S. L. Librach 7T0

As physicians, we are responsible for maintaining the health of our community. We spend years in medical school and in postgraduate training learning the signs and symptoms, the pathogenesis and the management of a multitude of conditions, many of which we will rarely encounter during our professional careers. We spend years digesting myriads of tidbits of information gained from research, much of which is of doubtful practical significance. Yet, we neglect almost entirely a condition which overall ranks a close third behind heart disease and cancer as a killer in our community. Accidents are important! More than 10,000 Canadians die each year as a result of accidents and the mortality rate continues to creep upward each year. However, it is not only the awesome and gruesome upward spiral of mortality rates which should capture our attention, but also the accompanying staggering morbidity rates. Accidents maim, cripple, disfigure, and disable a good number of our population (in 1951, 121,000 were permanently disabled due to accidents — 1% of the population of Canada at that time). In terms of loss of productivity and economic importance, accidents are more important than any other condition. Many of the strides taken in the investigation of accidents and in accident prevention have been achieved with very little cooperation from the majority of physicians. We tend to deal with the end results of accidents often without considering etiology, pathogenesis, and prophylaxis — a remarkable oversight by our usually obsessive profession. The time has come for physicians to take more active interest in understanding and preventing accidents.

Of course, one of the keys to understanding accidents and accident prevention is education. In our medical school, discussion of accidents and accident prevention is confined to and often hidden in three or four hours of lectures in the Public Health and Preventive Medicine course, lectures which unfortunately many of us skip or sleep through. We have on the whole an "unhealthy" attitude to Public Health, an attitude nurtured in part by the similar attitude of many clinicians and in part by the static departmental curriculum setup itself. It is to be hoped that the new curriculum has included in it a spirited discussion of accidents and accident prevention as an essential topic. However, such discussion and education should not end with graduation. The C.M.A. should in some way attempt an education programme for the seemingly unconcerned, uninformed and, possibly, misinformed majority of its members. As it stands now, most of the information we obtain on accidents is "accidental".

Not only must we be able to educate medical students and graduate physicians on the various aspects of accidents, we must also be able to pass this information along to our patients. Accident victims (those who survive in any case) become patients of physicians. Would it not be possible for a physician to counsel such patients on accident prevention just as he counsels patients with bronchitis to give up smoking? Tell them to wear seatbelts! Tell them to wear safety goggles at work! This is just a matter of prophylaxis, an integral part of the management of most conditions. Remember, accidents are caused and don't "just happen". One note of caution: practice what you preach. An obese doctor prescribing a diet for a similarly obese patient can hardly be effective.

Besides education, another key factor which could enable physicians to do something about our appalling accident statistics lies in an inherent characteristic of our profession. The medical profession forms one of the wealthiest, most respected and most influential groups in our society. Because of this, physicians should be able to bring considerable pressure to bear on our governments to promote new safety regulations and to provide more funds for accident research and accident education. In order to do this however, we must have coordinated action by a large number of concerned physicians. This goal has not been achieved yet.

In the past, the medical profession, unfortunately, has been relatively unconcerned about accidents and accident prevention. However, because of the growing importance of accidents as a community health problem, physicians must overcome this neglect. "The life you save, may be your own".

Head Trauma

F. J. H. Payne, 7T0

Part I: Classification, Mechanism and Pathology

Practically none of us have reached adult life without having sustained some degree of brain injury! Although infancy and early childhood are the most hazardous periods for the brain, head injury still ranks as the Number One Killer of people between the ages of one and 44.

Part I of this article deals with some aspects of *closed* craniocerebral trauma in the adult with particular reference to classification, clinical features, mechanisms and pathology. In Part II, the complications of closed head trauma will be reviewed.

CLASSIFICATION

Generally, the severity of cerebral injuries is directly related to the duration and degree of loss of consciousness. The following classification has been suggested.[1]

Brain Trauma I. (More than two thirds of patients)
- — unconsciousness lasting minutes
- — stupor or confusion lasting not more than a day
- — slight neurological findings lasting up to four days

Brain Trauma II. (20% of patients)
- — unconsciousness lasting one to 24 hours
- — confusion lasting up to four days
- — slight neurological findings lasting over four days

Brain Trauma III. (10% of patients)
- — unconsciousness lasting one to seven days
- — stupor, confusion or serious neurological findings lasting for more than four days

Brain Trauma IV. (1% of patients)
- — unconsciousness lasting weeks
- — neurological brain stem symptoms with partial inhibition of cerebral cortical function

The clinical picture seen more or less characteristically in some of the commonly occurring conditions frequently developing after injury in certain areas will be described. Such description depends in part on the severity of the injury.

CLINICAL FEATURES

Concussion

This is the commonest concomitant of a blow to the head. It can occur without evidence of contusion, fracture or hemorrhage. It is characteristically attended by instantaneous loss of consciousness (the essential feature), coma and paralysis, and usually by some degree of amnesia for events which occurred both prior to and following the injury. Retrograde amnesia is the hallmark of concussion. Although concussion is commonly followed by graded stages of recovery, it is not always a transient, completely reversible phenomenon, and can be permanent with residual symptoms. The longer the post-traumatic amnesia. the greater the likelihood of residual symptoms. With a mild concussion, the victim recovers quickly, passing through phases of stupor, restless confusion and automatism in a period of minutes. However, since the effects may be severe with long, continued disturbances of consciousness, it is best to recognize grades of concussion and to remember that death can occur from pure concussion alone. With severe concussion, the patient goes through the same phases seen in mild concussion, but in a prolonged, extenuated way. During the period of confusion or decortication, acalculia, or visual disorientation may be superimposed. There is a period in which the patient seems to act and talk fairly normally but is in a stage of automatism in which memories are not being stored. Afterwards, he retains no memory of that period. The final phase of subsiding cerebral impairment (the post-concussive state) is characterized by ready fatigue on mental effort, dizziness, irritability, and impaired concentration, emotional instability with headaches, commonly with associated fatigue and tension. In severe concussion, bilateral extensor plantar responses are usually found without other abnormal physical signs. The CSF is not necessarily under increased pressure. The time taken for recovery, and the presence or absence of residual symptoms, depends not on the nature of the injury, but on its extent and severity. Young people show better recovery. Cases with incomplete recovery usually have suffered contusion and hemorrhage as well, though probably some are the result of severe concussion alone.

Subarachnoid Hemorrhage

This is quite common with major head injury. Usually a cerebral contusion is present. The patient complains of a throbbing headache, a stiff neck, is irritable and photophobic. Twelve to twenty-four hours following injury, a positive Kernig's sign is present and the patient tends to lie on his side rather than on his back or abdomen.

"Wet Brain" or External Hydrocephalus

This usually occurs in a patient with subarachnoid hemorrhage. The headache tends to increase in severity some five to seven days after injury. Papilledema may

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develop and possibly sixth nerve palsies. Repeated fluid studies will reveal xanthochromatic fluid with high pressure. There are usually no localizing signs suggesting a unilateral hemispheral lesion.

Frontal Lobe Contusion

Clinically one finds prolonged confusion, prominent delirium and urinary incontinence which may occur even after the patient appears to have recovered consciousness. Memory disturbances and disorientation are also seen.

Temporal Lobe Contusion

These patients generally recover from coma early but are hemiparetic or hemiplegic, more stuporous than one would expect, and aphasic if the dominant hemisphere is involved. Frequently there is daily or hourly variance in the pupil size, and it may be difficult to differentiate contusion from edema due to a developing subdural or extradural hemorrhage.

Parietal Lobe Contusion

These injuries may cause hemiparesis or hemiplegia, and if the patient is sufficiently alert, homonymous hemianopia. If the major hemisphere is involved, aphasia, both receptive and expressive in nature, is present.

Occipital Lobe Contusion

This is relatively uncommon even though injury to this area of the skull is quite common. Hemianopic signs are seen. Most commonly, injury to the occipital skull causes olfactory disturbance with frontal and temporal lobe contusion by the contrecoup mechanism.

Cerebellar Contusion

This is unusual and generally if cerebellar signs such as nystagmus and ataxia are present, they are related to injury to the brain stem as well.

Brain Stem Injury

This is usually accompanied by diffuse cerebral hemispheral injury and is manifested by prolonged coma with no response to painful stimuli. Later it is followed by increased tone in the extremities. Decerebrate posturing and reflexes become more active early following the injury. There is a fluctuation in the blood pressure, pulse, temperature and respiration. Pupils are usually small and may be non-reactive, later irregular and unequal. Still later, sucking and chewing movements are noted.

Increased intracranial Pressure

Cerebral edema may follow any injury to the head and can be difficult to differentiate from extradural, subdural, subarachnoid or subcortical hemorrhage. Regardless of the cause of the increased intracranial pressure, the patient manifests a fairly constant pattern of progressive change. A deterioration in the level of consciousness is the most important sign to watch for. The

patient's ability to converse should be noted as well as his mental status. Other signs to look for are his behaviour, his response to command, the presence of pointless speech and of aimless movement, urinary incontinence and lastly, lack of response to severe physical stimulation (except by a quickening of respiration and spasms of extensor rigidity). In the early stages, there are headache, drowsiness and listfulness. With a rapidly expanding lesion, the pulse rate falls and is bounding. Blood pressure rises and the pulse pressure widens, due to bain stem pressure effects stimulating the vagal and vasopressor centres. The respiration slows and becomes stertorous. These changes do not occur if the vital centres are damaged. The same changes occur in acute cerebellar hemorrhage, or swelling, but the conscious level may not be impaired until death is imminent.

Neurologically, pulpillary changes occur when the third nerve is compressed. First the pupil may be smaller on the involved side but later enlarges from third nerve distortion by the herniated uncus. Then, ptosis of the lid develops and the eye may deviate outward. This is especially true in unilateral compression. Following this, increased reflexes are usually detectable on the involved side and upgoing toes develop in the contralateral foot; then spacticity of the limbs and finally decerebrate posturing. Paralysis of the opposite limbs and face occurs when the subdural and extradural hematomas are large enough to cause hemispheral ischemia from direct compression or brain stem involvement from the uncal herniation. Rarely, hemiparesis develops on the ipsilateral side ("crus syndrome"). Drowsiness increases to confusion, then to stupor with withdrawal movements to painful stimuli which are less on the ipsilateral side if the compression is unilateral. Finally, deep coma occurs with absent swallowing, cough, corneal and lid reflexes, non-reactive pupils and terminally, areflexia.

The Alcoholic

To differentiate clinically the effects of alcoholism from those of head injury, is frequently difficult. As a general rule, the intoxicated alcoholic will have poor reacting pupils. He is apt to be hypo-reflexic and to move all four extremities equally to painful stimuli. To differentiate a subarachnoid hemorrohage in a belligerent patient who also has contusion, from alcoholism, is difficult as well. The alcoholic, as a general rule, tends to be hypotensive. If there is no evidence of injury elsewhere, hypotension in a head injury is often suggestive of alcoholic intoxication. An alcoholic tends to vomit a little more than the sober, severely head-injured person.

During the acute stage of craniocerebral injury, hypertensive vascular reactions are far more common than hypotensive reactions. The more extensive the brain injury, and the more acute the increase of intracranial pressure, the more distinct and frequent is the hypertensive reaction. Real shock is rare except in cases with additional injuries of thorax, abdomen or extremities.

MECHANISM AND PATHOLOGY

The mechanisms leading to brain injury are grouped into blunt and penetrating head trauma. Blunt blows usually produce non-penetrating close injuries. The CNS changes resulting from blunt trauma are classified as:

1. Primary tissue alterations
   — direct result of mechanical effects
   — always focal and often multi-locular
2. Secondary alterations
   — result of cerebral or peripheral circulatory disturbances and post-traumatic brain hemorrhages with their complications.

Primary alterations include: a. Contusion of the leptomeninges and cerebral cortex; b. Primary hemorrhages into white matter; c. Epidural, subdural and subarach noid hemorrhage.

Disturbances of the circulatory system arise from: a. Direct irritation or damage of brain stem regulatory centres; b. Indirect irritation or damage due to primary tissue lesions in the cerebrum; c. Effects of peripheral lesions on the CNS independent of simultaneous central lesions (blood loss, hypoxia, circulatory collapse).

The three types of head-trauma caused by a blunt blow are: acceleration, impression and rotation trauma.

Acceleration Trauma

The most frequent violence suffered by the skull and brain is that of translation trauma, i.e. the head is accelerated with practically no rotation. At higher accelerations, this results from the so-called contrecoup lesions. This term is incorrect since the brain does not collide with the antipole but tries to get away from it. In a collision between the head and a large solid mass, the skull is abruptly decelerated as a whole but the inertia keeps the brain moving. This produces an over-pressure at the surface of the brain at the site of impact and an under-pressure (suction) at the side opposite the impact point. The over-pressure will not cause any cortical damage whereas the under-pressures does, being responsible for the contrecoup foci. The capillaries are destroyed at a certain magnitude of decreased pressure because gas bubbles are produced which cause vessel rupture. The bubbles may also develop in the tissues, which inflate and are thereby destroyed. The contrecoup injury can only be produced in an unsupported skull because only the freely moving head may be accelerated or decelerated and go through the above mechanism.

Impression Trauma

When the skull collides against a small object, it remains comparatively still. After the initial positive pres-

sure at the impact pole, a negative pressure is produced by the quick rebound of the depressed bone. As a result of the cavitation produced by the negatvie pressure, primary lesions occur at the collision point.

Therefore, cortical contusion (cortical cavitation trauma) may be the result of either acceleration or impression trauma and not the result of an assumed contusion of cortex against the skull. These lesions are usually particularly extensive at the base of the frontal and occipital lobes but also occur widely between the base and the brain convexity. Concomitant subarachnoid hemorrhages are a constant feature. If the antipole coincides with an area rich in CSF (i.e. a cistern) the developing cavitations have no effect on the fluid. This is the only situation in which the cushioning effect of CSF is justifiably applicable.

In most cases of a sagittal blow to the skull, the bitemporal axis is lengthened, the sagittal axis is compressed, the volume of the cranial cavity increased, and the ventricles dilated. Since the duration of the impact is too short for the CSF to fill the suddenly enlarged ventricular space, negative pressure and cavitation occur in the ventricular system in a way similar to that seen at the antipole. The ventricular walls and portions of corpus collosum placed beside the ventricles are the principal sites of primary traumatic hemorrhages. This explains the origin of central brain lesions.

Rotation Trauma

The best example of rotation trauma is a hook on the chin by the fist. In this rapid rotational movement of the head, the brain lags behind the skull, the inner surface of which rotates over the brain. A whirling motion is imparted to the tissues which move in relation to each other, producing destruction from sheering, stretching and compression forces. The nervous tissues are lacerated as neurons and connective veins are stretched and torn. The effects of a sheer strain depend on the severity of the sheering, the direction of distortion relative to tissue orientation (fibre tract direction) and the elasticity of the tissue elements. The pattern of rotational motion varies depending on the site and intensity of the blow, the moveability of the head and the degree of intracranial pressure already present.

A lesser degree of motion occurs in the frontal and temporal lobes which are confined in the more rigid anterior and middle fossae than in the parietal and occipal lobes. It is in this parieto-occipital region that maximal convolutional displacements (compression-rarefaction waves) occur. The greatest sheer strains most likely develop in the anterior frontal and temporal lobes. However, they also concentrate in the vicinity of the brain stem and craniospinal junction resulting in the neurocellular dysfunction producing concussion. For a substance like brain, which is extremely incompressible, yet shows little resistance to deformation of shape, sheer strains rather than compression rarefaction strains can be expected to be the prime cause of injury.

Concussion can be considered a rotational injury. Ambroise Paré, the great 16th century French surgeon, observed in his writings on brain concussion[2] that *many die of small wounds; and others recover of great, yea, very large and desperate ones, according to the variety and condition of the temperaments.* Four centuries later, our understanding of the "functional" versus structural aspects of concussion which Paré alluded to, still remains relatively superficial. Impaired function of a central reticular activating region is related with many of the phenomena of concussion. Two types of cellular depression can occur:

1. Prompt depression, due to the direct action of the force on the cell
2. Delayed depression, due to a chemical alteration which may possibly be an excessive accumulation of acetylcholine.

These changes may not be due to the direct effect of the force on the cell but may occur secondarily to damage sustained by the descending tracts of these cells by stretching the neck.

In its mildest form, cerebral concussion is probably due to traumatic depolarization or hyperpolarization of the neuron. However, a second mechanism may also operate. Since the brain is capable of handling incoming signals of only certain proportions, it is possible that if a sudden, massive inflow of impulses entered the reticular substance, normal handling would be disturbed and a physiologic as well as a mental confusion might develop. It is known that excessive stimulation of a neuron group can set up abnormal reverberating circuits which may persist for seconds to hours. This would lead to confusion or complete loss of consciousness yet be followed by complete recovery when the state abates. The syndrome of delayed concussion characterized by drowsiness, pallor, vomiting, and diaphoresis coming on several hours after injury, may be attributable to chemical alteration in the nerve cell such as excessive accumulation of Acetyl Choline. While free Acetyl Choline is never normally present in CFS, it appears in relatively large quantities following head injury. The higher the concentration, the more profound the coma and the more marked the EEG changes. Acetyl Choline in high concentration can block synaptic transmission by depolarization of the postsynapic membrane. Therefore, concussion can block the sensory evoked responses in the RAS by restricting the spread of activity within it.

Cerebral contusion, unlike concussion, is actually a bruising of the brain with petechial hemorrhage. The anatomic changes of cerebral contusion are described as dispersed, minute, intracerebral hemorrhages without gross disruption of brain continuity. These are generally

in much greater concentration in the white matter than in the cortex.

The diagnosis is established by finding blood or xanthochromia in the CSF and a focal abnormality in the EEG. If injury is confined to a "silent" area of cortex, and is not associated with concussion, the patient can be alert and asymptomatic.

Respiratory disturbances have been observed in association with a variety of intracranial abnormalities, such as:

1. Upper airway obstruction
2. Ventilatory abnormalities
3. Aspiration pneumonia (probably the most common extracephalic complication accompanying severe head injury.
4. Pulmonary edema

The mechanism of paroxysmal neurogenic pumonary edema following brain injury involves a systolic and diastolic overload of the left ventricle connected with a sympathetic stimulus. The same adrenal sympathetic response also causes inadequate left ventricular relaxation which rapidly elevates the pressure in the pulmonary capillaries. Increasing intracranial pressure triggers systemic venoconstriction, transient bradycardia, a positive myocardial inotropic response and severe systemic arteriolar vasoconstriction. The left ventricle may not be able to increase its output against a high peripheral resistance or relax sufficiently to accept an increased systemic venous return. Thus, the left atrial pressure may rise to extraordinary levels with flooding of the pulmonary vascular bed causing severe pulmonary edema.

Arterial hypoxemia has been observed in patients with localized head injuries in the apparent absence of any of the above disturbances. This response is due to pulmonary arteriovenous shunting induced either directly from the effects of mass sympathetic discharge on the pulmonary vasculature, or passively as a consequence of changes in pulmonary blood flow secondary to the systemic neurovascular response. A determination of arterial oxygen tension in patients with intracranial pathology is of critical importance.

Cardiac disturbances have been associated with subarachnoid hemorrhage, cerebral infarction, meningitis, status epilepticus, and intracranial mass lesions. ECG abnormalities are seen most consistently with subarachnoid and intracerebral hemorrhages. The commonest abnormalities are: 1) *Depression or elevation of ST segments;* 2) *Prolongation of the Q-T Interval and* 3) *Inversion of the T wave.*

The ECG may show arrhythmias or display the characteristic picture of an acute myocardial infarction. The increased intracranial pressure may promote cardiovascular changes by acting on the parasympathetic and sympathetic nerve centres in the hypothalamic region governing cardiovascular activity. The encephalogenic heart lesions are probably brought about by acute intoxication with endogenous catecholamines (probably norepinehprine) released from adrenergic cardiac nerve.

Part II: Complications

I. Intracranial Hypertension & Edema

Following cranial cerebral trauma, intracranial hypertension may be due to edema, hemorrhage or cerebral vascular congestion. The marked changes in intracranial pressure which may be sufficient to cause death are most likely due to rapid alterations in cerebral intravascular blood volume. Cerebral swelling following head injury, has been attributed to edema, internal hydrocephalus, diffuse extravasation of blood and cerebral-vascular congestion. At least two factors are known to operate in the development of acute swelling.

Impaired tone may be due to: local oxygen want (the most likely cause of vasomotor paralysis); vascular overdistension; mechanical distortion of a specific hypothalamic centre producing a vasoparalysis resulting in vascular distention and noncomitant rise in intracranial pressure, a form of cerebral swelling. Loss of cerebral vasoconstrictor tone appears to be the most likely cause of brain swelling and accounts for the initial increase in intracranial pressure.

If loss of vasomotor function is transient, and if adequate respiration is resumed, the pressure gradually returns to normal. With prolonged apnea, the vascular effects of CO_2 retention and hypoxia cause further dilation and intracranial pressure continues to rise. Therefore, the increased intravascular blood volume secondary to vasal dilatation and subsequent vasomotor paresis contribute to the brain swelling responsible for the intracranial hypertension. This volume change is probably always generalized. The systemic blood pressure rise with the increasing intracranial pressure, presumably nature's effort to offset the resultant ischemia, is known as the Cushing phenomenon. However, the paralyzed vascular tree is flooded thereby increasing the intracranial pressure.

The vascular responses productive of cerebral swelling are differentiated from the changes developing in

brain tissue itself, properly referred to as brain edema, although such circulatory changes undoubtedly facilitate the development of edema. Its pathogenesis is unknown. Edema begins within minutes after concussion and progresses to a maximum in roughly seventy-two hours. Brain edema may result either from primary brain damage or more usually, secondarily from brain compression by clots. Edema associated with various types of brain injury exhibits a predilection for the interstitial spaces of the subcortical white matter whereas edema consequent to circulatory arrest involves intracellular swelling in cortical areas. The greatest danger comes when asymmetrical lesions develop, such as the edema which forms under an epidural or subdural clot. The pathogenesis of cerebral edema in the gray matter is different from that in the white matter. Although the extra cellular space increases in both areas, its is primary in the white matter and secondary to cellular rupture in the gray matter. Edema may occur alone but commonly is the result of brain laceration, particularly in the region of the temporal lobe tip and under-surface of the frontal lobe.

II. *Hematomas*

Probably between 1%-2% of all extradural and subdural hematomas lie in the posterior fossa. Their clinical pictures seem identical. In the less extensive and often unilateral posterior fossa hematomas, upward herniation of the cerebellum tends to be delayed and minimal so that hydrocephalus is a late feature. Although there is distortion of brain stem vessels, they are not stretched and vascular tears and hemorrhages tend to occur later. The caudal part of the RAS is affected, diminishing its drive on the rostal RAS, causing a trance-like state of stupor, closely resembling normal sleep. The infratentorial hematoma pushes the pons and medulla forward and laterally causing mesencephalic tegmental distortion. This is commonly evidenced by ptosis and loss or diminution of upward gaze, often most marked on the side of the clot. The tonsils are herniated into the foramen magnum probably accounting for the frequent finding of nuchal rigidity. Infratentorial hematomas primarily produce profound autonomic disorders because of the medullary pontine and mesencephalic distortion.

In contrast, supratentorial hematomas are characterized by rapid deterioration of consciousness, usually in deep coma with decerbration and Cheyne-Stokes respiration. The rapid loss of consciousness is probably due to acute lateral and longitudinal distortion of the rostral (diencephalic) RAS.

Epidural hematomas usually occur on the side of the head that receives the blow, and most frequently in the parietemporal region. About 80% are located in the middle fossa usually in the temporal region along the course of the middle meningeal artery and vein, and so are arterial or venous in origin (usually arterial). They characteristically develop quickly. The mortality rate of nearly 100% in untreated cases is due to the rapid shift of the brain with subsequent brain stem compression. They are the most fatal of the head injury complications and cause death unless removed. The classical pattern, with a temporal clot, of initial loss of consciousness followed by an asymptomatic "lucid interval" and this in turn by signs and symptoms of increased intracranial pressure, occurs in less than 40% of cases. Frontal extradural hematomas are suspected if the patient complains of increasing bifrontal and retroorbital pain and headache. More slowly developing hematomas produce signs of frontal lobe dysfunction, namely, impairment of memory, concentration, judgment; intermittent disorientation, unpredictable actions, confusion and incontinence. Lumbar puncture often reveals xanthochronic fluid under slightly increased pressure. But, in general, the clinical picture is indistinguishable from that of middle fossa hematomata. Posterior fossa hematomas have no characteristic clinical course, but may show muchal rigidity, nystagmus, a tendency to lie on one side and generalized hyporeflexia and hypotonia.

Subdural hematomas occur more frequently than the epidural and tend to spread more thinly and widely. They are usually of venous origin.

a. Acute subdural hematomata are usually due to laceration of one or more of the connecting veins between the cortical surface and sinuses. They commonly occur on the contrecoup of the skull. Symptoms usually develop within the first few days of injury and consist chiefly of deepening coma and a decerebrate state.

b. Subacute forms may produce symptoms a week or more after injury (headache, personality change, dulling of sensorium). They tend to come and go. Within a few hours or days, the hematoma breaks down, forming a high osmotic pressure fluid that becomes enclosed within a semi-permeable membrane. Progressive increase in volume results from the increased osmotic tension.

c. Chronic subdural hematomas are more likely to result from blows to the forehead or occiput than from blows to the side of the head. Most occur in infants and elderly patients, the high susceptibility in the elderly being due to increased width in the subarachnoid space secondary to cortical atrophy. Characteristically weeks or months pass before the insidious appearance of symptoms which include:

   i. Headache (probably the most conspicuous early complaint)
   ii. Personality change (one of the most common early symptoms)

iii. Gradual inattention, indifference, and lethargy
iv. Impaired judgment
v. Occasionally delusions, convulsions and, with left-sided lesions, aphasia

Intracerebral hematomas are the rarest of the mass lesions complicating head injury. They usually occur subcortically along the orbital surface of the frontal lobe and in the temporal lobe tips, are usually multiple and form fairly rapidly. In a month or two the clot is converted into a closed space filled with serum. They present, acutely or chronically, in a variety of ways, being frequently associated with subdural hematomas. Usually there is a deepening level of consciousness, frequently hemiparesis, and often seizures.

III. Post-Concussive Syndrome

Unfortunately this is an erroneous term for it leads to the inclusion of pure anxiety neuroses, hysterical states and compensation reactions in a manner focussing attention unreasonably on minor head injuries.

It is the commonest sequel of craniocerebral trauma. It is characterized by headache, vertigo, nervousness, irritability, insomnia, memory impairment, inattention, excessive fatigue and alcohol intolerance. Development of these symptoms is not clearly related to the severity of the injury. Signs occurring in the later stages of recovery clear up completely or nearly so, but may take up to two years.

IV. Headache

Traumatic, casually-related, organic headaches rarely persist after a head injury. Most persistent headaches are psychogenic and occur more commonly after mild injury. Headache which increases weeks after trauma is the commonest indication of subdural hematoma.

V. Dizziness

Transient postural vertigo, occurring briefly with a quick head movement, is seen in about 25% of head injury cases (without temporal bone fracture). It usually persists several months and is not usually associated with cochlear symptoms.

VI. Epilepsy

There is a 1% risk of epilepsy in colsed injuries without a fracture or hematoma. The clear relation existing between tissue destruction and the development of fits does not exist between the severity of damage and the likelihood that the patient will stop having fits. Antecedent conditions to be ruled out include old idiopathic epilepsy and symptomatic epilepsy related to a non-traumatic lesion which had run a silent course, but had become manifest from craniocerebral trauma or shock (vascular maformatoins which start bleeding).

VII. Focal Cerebral & Cranial Nerve Lesions

Degrees of dysphasia and visual field loss are commonly missed. The cranial nerve most often affected is the first. Although the ccrebellum is seldom damaged in closed injuries, severe ataxia and tremors may result from midbrain hemorrhage.

INVESTIGATION

a. Skull x-ray may reveal shift of a calcified pineal gland or choroid plexus.

b. EEG is of great value if done in the early stages and repeated later to show changing electrical abnormalities.

c. Echoencephalography is used to detect midline displacements. Its 95% accuracy is reflected in the frequent detection of subdurals and less commonly extradurals. However, with bilateral subdurals there may be no shift and even sizeable surface clots may not shift the midline structures in the first few hours of injury.

d. Percutaneous common cartoid angiography usually shows epidural, subdural or intracerebral hematoma. Without angiography, about 50% of the hematomas cannot be recognized from clinical evidence alone because approximately half of these do not present with the classical symptoms.

e. Ventriculograms or subdural air injection.

f. Brain Scan, when repeated, may prove informative in the more slowly evolving lesions following trauma.

g. Rheoencephalography is the only method which permits continuous indirect observation of intracranial circulatory changes. It is the best method for differentiating post-traumatic brain edema from brain compression due to hematoma, abscess or air accumulation.

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Chest Trauma

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On the North American continent, trauma is the third leading cause of death, preceded only by cardiovascular disease and cancer. Automobiles are largely responsible for this fact, and of the 50,000 people killed in the United States in 1968 in automobile accidents, one quarter died as a result of chest injuries. Because of the increasing number of such injuries in our accelerating society, the physician must be adept at the diagnosis and management of the common chest injuries and be familiar with, and watchful for, the less common.

Chest injuries can be classified broadly into two groups: those due to blunt trauma and those due to penetrating agents.

BLUNT TRAUMA

ETIOLOGY

In the United States, fifty percent of the cases of blunt chest trauma are due to automobiles. Falls and assaults account for the other 50%. In this country however, automotive injuries account for an even greater percentage (approximately 80%), while assaults are a much less significant cause of injury. Of those sustaining chest injuries in auto accidents, one half are drivers and the other half are either passengers or pedestrians in approximately equal numbers.

TYPES OF INJURIES AND MANAGEMENT

(a) *Rib Fractures*

There are two types of rib fractures:

1. those that occur at the site of injury with the fragments displaced inwards, and
2. those resulting from antero-posterior compression of the chest with a 'bursting out' at the site of maximum convexity (the angle of the rib) resulting in outward displacement.

The most frequently involved ribs are the 6th, 7th, and 8th. The physician should bear in mind that the greater the number of ribs fractured, the greater the likelihood of intrathoracic injuries. This does not apply however to children under 5 years of age as they frequently have severe intrathoracic injury with no rib fractures.

Ribs require relatively little treatment when fractured as they are usually well aligned and minor degrees of malalignment are of no clinical consequence. No treatment may be necessary; however, the repetitious pain created at the fracture site by respiratory movement, aggravated by coughing and deep respiration results in both voluntary and involuntary muscle splinting of the thoracic wall. If the degree of discomfort and impairment of respiratory function is significant then intercostal nerve block is the best treatment. This is done in the paravertebral area, lateral to the mass of the paraspinal muscles. 2-3 ml. of 1-2% Xylocaine are injected under the involved ribs into the intercostal nerve area. The injection will relieve pain for several hours (1-12) and can be repeated. The nerve block allows the patient to cough with little or no pain and prevents atelectasis.

Circumferential adhesive strapping of the involved side of the chest is still used successfully by some physicians, but it tends to prevent adequate expectoration of secretions, resulting in atelectasis and possibly infection.

(b) *Fracture of the Sternum*

Undisplaced fractures of the sternum require little or no treatment. If the fracture ends are overriding however, they must be reapposed surgically and the position maintained with wire sutures. Other injuries may be associated with sternal fractures depending on the mechanism of injury. The anatomical proximity of the mediastinal structures should make the physician consider the possibility of pericardial effusion, hemopericardium, contusion of the heart muscle and damage to the coronary arteries, if the patient received a direct blow to the chest. He should also be aware that a sudden, severe flexion of the thoracic spine as may occur in a mining accident may result in both a fractured sternum and compression fracture of the spine.

(c) *Flail Chest*

The characteristic steering wheel injury typically results in fractures of the 6th, 7th and 8th ribs on the left with frequent disruption of the costochondral junctions. Due to the relative lack of supporting musculature, this type of injury is likely to produce a flail chest.

Diagnosis is always made from physical examination. The patient is dyspneic, restless and frequently uncooperative. He may be cyanotic, tiring rapidly and close to respiratory arrest. Because the flail area is essentially detached from the rest of the rib cage, it is sucked in by the negative intrapleural pressure on inspiration and pushed outward on expiration — a phenomenon referred to as paradoxical respiration.

X-ray examination will usually demonstrate the fractured ribs but disruption of the costal cartilages which often act as the medial or lateral border of the unstable segment cannot be detected radiologically. A central flail segment including the sternum may result from bilateral fractures through costal cartilages and in this case, no rib fractures are seen on x-ray examination.

In a series by J. H. Duff et al., the diagnosis of flail chest was delayed in approximately 16% of cases and the mortality of this group was significantly increased. Many of these patients had no obvious paradoxical respiration on admission, but several hours or days later, when secretions had accumulated, the increased muscular activity required to inflate the lungs made the flail progressively more apparent.

The principal biochemical abnormality in the patient with flail chest is hypoxemia. Arterial pCO_2 however, is normal or decreased indicating that there is adequate alveolar ventilation (Whitman and Norman). The larger the flail segment, the greater the effort required to produce adequate pulmonary ventilation. Hypoxia results from the increased oxygen requirement necessary to meet the energy demands.

Although small flail areas may be treated by immobilization with a pressure dressing, large areas with paradoxical movement require aggressive therapy. Unstable ribs or sternum may be held outwards by skeletal traction, but usually a cuffed endotracheal tube is inserted into the patient's airway and connected to a positive-pressure, patient-triggered respirator. Within 48 hours an elective tracheostomy should be performed. This form of treatment provides a form of internal splinting, helps to prevent pulmonary edema which may follow 24-48 hours after injury, and allows repeated bronchial suction which further reduces the chance of pulmonary edema and atelectasis. After 14-21 days the chest is sufficiently stable to allow gradual weaning of the patient from the respirator.

(d) *Pneumothorax*

Air in the pleural space results from either open or closed injury to the lung. Open pneumothorax, in which air enters the pleural space through an external wound will be discussed under penetrating trauma. Closed pneumothorax results from air leaking from the lung into the pleural space, either through a rent in the visceral pleura, the trachea, or a major bronchus. The interference with ventilation produced by closed pneumothorax depends upon the degree of pulmonary collapse and the pulmonary reserve. With lesser degrees of collapse, there may be few or no symptoms. With greater collapse, there will be severe dyspnea and other symptoms, especially if pulmonary reserve is limited. On examination, hyperresonance to percussion and decreased breath sounds will be heard over the affected lung. There may be a shift of the trachea towards the *uninjured* side, although there is usually no shift.

In the absence of symptoms, pneumothorax with less than 10% collapse is usually treated expectantly, with frequent clinical and x-ray evaluation. The exception to this however, is the patient with a small pneumothorax who must be intubated and operated on for other injuries. In this case, pleural catheter drainage *must* be used. The catheter is inserted in the second intercostal space usually just lateral to the lateral edge of the pectoralis major muscle. In most large centers, the catheter is connected to continuous suction with a pump capable of removing large volumes of air. Alternatively, water-seal drainage may be used. If atelectasis persists despite adequate thoracic drainage, bronchial aspiration or bronchial suction through a bronchoscope should be performed.

Tension pneumothorax is a potentially fatal condition that must be treated immediately by decompression. In an emergency situation outside the hospital, a needle of any size, the larger the better, should be inserted into the pleural space. In the hospital a large rubber tube should be inserted into the pleural cavity through a trocar.

The patient with tension pneumothorax usually presents with cyanosis, dyspnea, tachypnea, arterial hypotension and impaired cerebration from hypoxia. The involved side of the chest moves poorly with respiration, and is hyperresonant. Breath sounds are diminished. Neck veins are distended and the trachea is shifted toward the uninvolved side.

The most popular explanations for the lethal potential of tension pneumothorax are based on an assumed interference with the venous return to the heart. Loss of negative intrathoracic pressure, compression of the venae cavae by positive intrapleural pressure and angulation of the caval-atrial junction secondary to mediastinal shift are the commonly accepted reasons for the supposed decrease in venous return.

Recent research (Rutherford et al.) suggests that these explanations may not be applicable in man. These studies on the goat — an an.imal whose mediastinum corresponds closely to that of the human in mobility and other features — could not demonstrate any evidence of caval-atrial obstruction in experimentally induced tension pneumothorax. They suggest that the pathophysiology is due to increasing degrees of pulmonary vascular shunting assocaited with parenchymal collapse. The failure of initial compensation for the mechanical interference with ventilation is presumably a result of cerebral hypoxia.

(e) *Hemothorax*

Hemothorax, a common sequel of chest trauma, is due to bleeding from either damaged pulmonary vessels, torn intercostal vessels or occasionally injury to the heart and great vessels. Clinically the diagnosis may be made by detecting decreased breath sounds and dullness to percussion posteroinferiorly in the patient capable of sitting upright. More commonly, in the severely injured patient lying supine, the presence of blood in the pleural cavity will not be detected until a radiological examination is done. However, if massive bleeding into the chest is suspected as the cause for arterial hypotension, diagnostic thoracentesis should be carried out immediately.

A minor degree of hemothorax is treated expectantly at first. Repeated clinical and radiological chest examinations will identify continued bleeding. If more than a minimal hemothorax is present a large rubber drain should be inserted under local anaesthesia into the 4th or 5th intercostal space in the midaxillary line. A lower level is contraindicated due to the risk of penetrating a high-lying diaphragm with injury to the spleen or liver. The drain is then connected to suction.

The advantages of this method of treatment are numerous:

1. Hemorrhage from small pulmonary vessels will usually stop rapidly with the re-expansion of the lung.

2. Rapid obliteration of the pleural space reduces the chance of empyema developing and also eliminates the need for later decortication procedures, necessary in the past for treatment of fibrothorax.

3. The drainage tube provides a good way to evaluate the presence and amount of continuing hemorrhage. In the presence of alarming hemorrhage, thoracotomy may be necessary to control the bleeding.

(f) *Mediastinal Injuries*

(i) Heart

Cardiac Tamponade

Free bleeding into the pericardial sac classically results in Beck's triad of signs of cardiac tamponade — distant heart tones, falling blood pressure and elevated venous pressure. Cardiac output is reduced in tamponade due to the decreased diastolic expansion of the heart and is probably not due to obstruction to the venous inflow at the entrance of the great veins as is generally stated in textbooks.

Pericardial aspiration is urgent and can be lifesaving. As little as 200 ml. of blood in the pericardial sac can cause death. Therefore initial management is pericardiocentesis for both diagnostic and therapeutic reasons. A subxiphoid approach with a No. 19 needle can be used, or alternatively, the needle can be inserted through the 4th intercostal space anteriorly just lateral to the sternum.

Clotted blood is responsible for tamponade in a large percentage of cases and is associated with a high incidence of false negative pericardiocenteses. Therefore, operation is advisable in those patients with a negative pericardiocentesis when circulatory deterioration intervenes.

Myocardial Injury

The true incidence of myocardial injury is difficult to evaluate and the type of injury ranges from mild myocardial contusion with minor EGG changes to massive rupture of the heart with sudden death. In the middle of this spectrum are a number of intracardiac lesions that are compatible with limited survival. Included in this group are ruptured aortic valves, ruptured intraventricular septa and rarely, avulsed papillary muscles producing acute mitral or tricuspid insufficiency. With the availability of modern operative techniques such as cardiopulmonary bypass, surgical correction of these lesions is now possible.

(ii) Injury to the Great Vessels

The thoracic aorta is cushioned laterally by each lung but in severe crushing injuries, its position between the sternum and thoracic vertebrae makes it susceptible to injury with complete rupture and almost instant death.

Accidents involving sudden deceleration can also produce traumatic rupture of the thoracic aorta. Anatomically the thoracic aorta is immobilized at three places — at its base, at the ligamentum arteriosum, and at the diaphragmatic opening. If rapid linear deceleration occurs, the more mobile sections of the thoracic aorta herniate anteriorly while traumatic traction forces are exerted at the fixation points and at the sites of origin of the major branches. The extent of the injury varies from intimal laceration to complete transection, but surprisingly, there is no significant correlation between the extent of the rupture and the length of survival. In one series by Parmley, 40% of the patients with *complete* transection of the aorta survived five days or longer.

Early routine chest films may show no abnormality, but subsequent films will demonstrate mediastinal widening, blurring of the outline of the aortic arch and fluid in the left hermithorax. Significant hypertension developing shortly after trauma in a previously normotensive individual is believed to be an important confirmatory finding. Other suggestive findings include:

1. Systolic or continuous murmur over the precordium.
2. Transient superior vena caval obstruction.
3. Significant difference in peripheral pulsations.

Aortography, in most cases will confirm or disprove the diagnosis and localize the site and extent of the rupture. Immediate surgical repair is undertaken preferably with direct end-to-end anastamosis. If this is impossible, a Teflon graft is inserted to restore aortic continuity.

(g) *Pulmonary Contusion*

Pulmonary contusion is generally considered to be a relatively minor, uncommon injury, and is often ignored in monographs on trauma. The truth of the matter is that pulmonary contusion is neither trivial nor rare.

The main reason for the lack of recognition of pulmonary contusion is the fact that this lesion is frequently associated with other serious, more apparent injuries,

and in the face of these injuries, the lung bruise may not even be considered.

Suspicion of its presence is the greatest aid in diagnosis. Rapid deceleration associated with automobile accidents, shock waves striking the chest wall from blasts and high velocity gunshot wounds, are the mechanisms most likely to give rise to severe pulmonary contusion. The removal of an associated hemothorax or pneumothorax may be required to reveal the full extent of the contusion which usually takes 4-24 hours to reach maximal size. The chest x-rays demonstrate an irregular infiltrate in the involved parenchyma.

Tachypnea, dyspnea and tachycardia are invariably present clinically, and the patient may have hemoptstysis.

The process of pathologic changes in the lung is constant. In the first 4-6 hours, bleeding occurs followed successively by stages of coagulation, absorption, and repair. The interstitial and intra-alveolar hemorrhage and edema result in parenchymal rigidity, and diminished gaseous diffusion, making the bruised lung incapable of normal ventilation. Survival depends largely upon the extent of injury and the respiratory reserve of the patient.

Treatment consists first of all in the management of any associated life-threatening lesions. Those being attended to, the use of a cuffed endotracheal tube and intermittent positive-pressure breathing apparatus is the best single therapeutic measure. The large quantities of bloody edema fluid and mucous can be removed by bronchial suction, and an airway assured. In 48-72 hours, an elective tracheostomy may be necessary.

Prophylactic broad spectrum antibiotics should be administered, and some researchers (Demuth et al.) recommend the use of steroids with the aim of reducing secretions and edema.

PENETRATING TRAUMA

ETIOLOGY

In times of war, penetrating injuries of the chest are frequently seen, while stab wounds and gunshot wounds are occasionally seen in the downtown hospital's emergency room.

TYPES OF INJURIES AND MANAGEMENT

Any structure may be damaged in a penetrating injury. Minor injuries such as those inflicted by knife blades may cause a small puncture and hemothorax, but there is usually minimal damage to deeper structures. Major damage to lung and mediastinum most commonly results from war wounds and gunshot blasts at close range. An open or tension pneumothorax may be present, foreign bodies, such as clothing and buck shot may be retained in the wound and the diaphragm may be ruptured.

(a) *Open Pneumothorax*

If the resistance to air flow through the opening in the chest wall is less than the resistance through the upper air passages, the lung will collapse completely on the side of the wound.

Therefore, it is urgent that open chest wounds be plugged. At the scene of an accident, blood soaked clothing, or the palm of one's hand can be pressed over a 'sucking' wound, while in the emergency room, the chest wound can be plugged with vaseline gauze as the patient performs a Valsalva manoeuvre. Subsequently a large bore rubber drain and suction will be required once the chest wall has been repaired.

(b) *Rupture of the Diaphragm*

The possibility of traumatic rupture of the diaphragm should be kept in mind after a penetrating injury, and the pathway of any missile reconstructed to see if a hernia could have occurred. Diminished excursion of the thorax, impaired resonance, cardiac displacement, and changeable, adventitious sounds in the chest suggest the diagnosis clinically. X-rays of the chest will usually confirm the diagnosis but when negative, especially after a deep stab wound, serial films are required to detect late herniation. Once discovered, the defect is repaired surgically, usually using a transthoracic approach.

(c) *Other Injuries*

Exploratory thoracotomy is frequently necessary in severe penetrating injuries in order to determine what structures have been involved and to locate bleeding sites and foreign bodies.

SUMMARY OF THE GENERAL PRINCIPLES OF MANAGEMENT

The fundamental problem in all major thoracic trauma is to correct the disordered chest function. By strictly observing the following principles of management, many critically injured patients can be saved.

1. Secure the airway and a dminister oxygen if necessary.
2. Relieve intrapleural tension.
3. Relieve pain.
4. Stabilize the chest wall.
5. Give blood as required.
6. Explore the chest when indicated.

With these principles in mind, a comprehensive plan for managing major thoracic trauma can be developed.

A. At the scene of the accident

1. Clear the airway of any obstruction to breathing and use an oropharyngeal airway if required.
2. Use mouth to mouth artificial respiration when necessary.
3. Stop bleeding of wounds by applying pressure.
4. Plug open (sucking) chest wounds.
5. Aspirate a tension pneumothorax.

B. In the emergency department

1. Intubate the trachea with a cuffed endotracheal tube and 'bag' the patient for adequate ventilation.
2. Aspirate or drain the pleural cavities if necessary.
3. Insert an intravenous catheter and take blood for cross-matching for transfusion.
4. If the patient is in shock, start saline and/or dextran immediately.
5. When available, pump in matched blood at the rate of 500 ml. every 5 minutes until the systolic pressure reaches 100 mm Hg. provided central venous pressure remains under 10 cm. of saline.

C. In the operating room

1. Insert a cuffed endotracheal tube through the mouth and ventilate with an intermittent positive pressure respirator where needed.
2. Insert pleural drains if necessary and connect to suction.
3. Perform a thoracotomy for continued pleural or pericardial bleeding, or for other injuries as indicated.

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The Nature Of Shock

Charles Omole, B.Sc., 7T1

The signs and symptoms of shock are self-evident but the nature of this clinical syndrome has been quite elusive. Lack of basic knowledge of this disorder has resulted in much controversy about its management. Since treatment of shock has to be instituted without much delay, an understanding of the pathophysiological and chemical mechanisms will be very helpful. This summary, therefore, will focus mainly on these mechanisms so that the signs and symptoms of shock may be better understood.

There is no universally accepted definition of shock. Each of the definitions in the literature tends to describe a certain aspect of the shock process. However, a more modern definition has recently been devised by Simeone[1]. According to this view, shock is "a clinical condition characterized by signs and symptoms which arise when the cardiac out-put is insufficient to fill the arterial tree with blood under sufficient pressure to provide organs and tissues with adequae blood flow." Whatever the definition of shock, it always consists of at least two parts: an external or internal destructive force and the body's response to this event.

Shock may result from failure of one or more of four separate but interrelated functions. These are:—

(1) the pump (heart)
(2) the fluid which is pumped (blood)
(3) arterial resistance vessels (arteriole tone)
(4) the capacity of venous bed (capacitance vessels or venous tone)

In the context of Blalock's etiologic classification these functions may be correlated (2):

1. Cardiogenic Shock: This implies failure of the heart as a pump and may be brought about by:
   a) 1) Myocardial infarction
      2) Serious cardiac arrhythmias
   b) Miscellaneous causes would include mechanical venous obstruction such as those occuring in the mediastinum with:
      1) Tension pneumothorax
      2) Shift of the mediastinum
      3) Embolus in the vena cava or vena cava ligation
      4) Cardiac tamponade
2. Reduction in the fluid which is pumped (blood volume).

This loss of volume may be in the form of loss of whole blood, plasma, extracellular fluid in the extravascular space, or a combination of these. This pathogenesis of hypovolemic hypotension was categorized by Allen as shown in Table 1.[2]

3. Changes in resistance vessels maybe brought about by specific disorders which include:
   a) Decrease in resistance
      1) Spinal anesthesia
      2) Neurogenic reflexes as in acute gastric dilation.
      3) Possibly the end stages of hypovolemic shock.
   b) Septic shock
      1) Change in peripheral resistance.
      2) Change in venous capacitance.

Table I.
PATHOGENESIS OF HYPOVOLEMIC HYPOTENSION.*

A. *Blood Loss*
   1) External – as in lacerations, bleeding ulcers, etc.
   2) Internal – as in femoral fracture, crushing injury, ruptured spleen, etc.

B. *Plasma Loss*
   1) External – as in burns where blood also may be lost with loss of plasma into the tissues.
   2) Internal – as in peritonitis.
   3) Crushing injury (where generally more blood is lost than plasma).

C. *Water Loss*
   1) Water deprivation – Electrolytes are concentrated and not lost.
   (The therapy indicated is water without salt).
   2) Water loss – as from vomiting, high intestinal pancreatic or biliary fistula, or diarrhea. (Since electrolytes are usually lost, the therapy indicated is water with electrolytes).

* From J. G. Allen in H. N. Harkins: Surgery: Principles and Practice (Philadelphia: J. P. Lippincott Company 1961. pp. 104-125).

CHANGES IN SHOCK

"Anoxia not only stops the machinery, it wrecks it"
— Haldane.

Shock of all forms appears to be invariably related to inadequate perfusion of certain organs and tissues particularly at the micro-circulatory level. Many of the changes that eventually develop are due to the resultant hypoxia. Since the changes at the cellular level begin before the appearance of the clinical signs, a consideration of the cellular changes is essential.

Cellular Changes

One of the earliest cellular changes of injury from whatever cause is mitochondrial swelling and, presumably, damage. Within the mitochondria, substrates are

normally oxidized with the production of high energy phosphate bonds in the form of ATP. The bulk of energy required for most of the endothermic biological processes is supplied by ATP and creatine phosphate.

In shock, the hypoxic condition changes the respiration of the cell from aerobic to anaerobic. This change results in a poor energy yield and a host of dependent processes are impaired. Of the dependent processes one of special importance is that energizing the Na-K pump with the result that Na+ leaks into the cell and the level of K+ in the blood rises progressively. Other processes affected include glycolysis, fatty acid utilization and protein synthesis. These account in part for the raised serum levels of glucose, lipids, inorganic phosphorus, and urea, which are often observed in the early phase of shock.

The damaged cells release substances closely studied for their part in the causation of shock. Many enzymes which leak from the cells are obvious candidates. Their effects may be directly harmful; they may activate plasma constituents which are potentially harmful. These enzymes may be responsible for the conversion of fibrinogen to fibrin which is an observed fact and which has been accorded a central role in the cause of shock by intravascular coagulation[3].

Other substances released affect the vasculature to various degrees. These will be considered in detail later.

Metabolic Acidosis

A consequence of cellular hypoxia

With prolonged cellular hypoxia, lactic acid accumulates. Lippmann[3] has pointed out that a breakdown of Pasteur effect, the inhibition of anaerobic glycolysis and lactic acid production by oxygen, could also result in a relative increase in lactate. This lactic acid accumulation is thought to reflect the extent of cellular hypoxia. The kidney as well as the liver and skeletal musculature have been shown to exhibit a net regional lactate production in shock. In addition, occult regional lactate production and lactic acidosis can occur in the absence of hypotension.

The hypocapnia associated with early shock frequently changes to hypercapnia in the late stages as the respiratory mechanism fails.

Initially, there is an increase in the rate and depth of respiration which tends to preserve the pH within the normal range of 7.35 - 7.45. Eventually, this compensatory mechanism fails as the blood bicarbonate continues to de displaced by the accumulating lactic acid.

The hyperpnea of shock is an important compensatory mechanism and is often a good clinical index of metabolic acidosis and hence cellular hypoxia. This phenomenon may be misleading because gram-negative sepsis, even in the absence of metabolic acidosis is occasionally associated with hyperventilation; this is of a central origin[4]. Attempts to correct this metabolic acidosis of septic origin with bicarbonate may cause further deterioration of the patient.

Microcirculatory Changes

The circulatory changes have been investigated thoroughly with respect to arterial blood pressure, heart rate, cardiac performance and other measures of the central macro-circulation. The microcirculation, by contrast, has received much less attention. This is paradoxical because much of the blood is within the microcirculation and all of the blood-tissue exchanges take place within the capillaries and the sinusoids.

The microcirculation may be regarded as the exchange system between the blood and the tissues. However, the exchange actually take place across the capillaries.

The exchange system is composed of interlacing, mesh-like, biological tubes — arterioles, capillaries and venules. The building blocks of this system of tubes are cells. The capillaries are composed of endothelial cells with no muscle coats as opposed to arterioles and venules with muscular elements in their walls. Sphincters control the capillaries at both the upstream and the downstream ends. These sphincters are the last smooth muscular elements along the branches of the terminal metarterioles.

These sphincters are sensitive to stimulation by the autonomic nervous system and by local metabolites. This sensitviity is such that the pre-capillary sphincters respond more fully than the post-capillary ones when subjected to the same degree of stimulus.

The flow of blood through the capillaries is intermittent and occurs through the activity of the sphincters which are also controlled to a certain extent by anoxia. Many substances diffuse back and forth through the pores between the endothelial cells. The passage rate through these pores depends partly on hindrance due to their length, complexity and electric charge. These factors are governed by the endothelial cells which can swell or alter shape and which presumably suffer the changes affecting other cells after injury or shock.

During shock, the cells forming the building blocks of the microcirculation swell and the internal structures are disrupted. Many diffusible substances are released from the damaged cells and these substances have regulatory effects on the vasculature both locally and systematically.

CHEMICAL AGENTS AFFECTING THE MICROCIRCULATION

The chemical substances released during shock include catecholamines, histamine, serotonin, polypeptides, etc. The concentrations of these substances in the circulating plasma result from the balance between the

rate of production and the rate of metabolism and excretion. The concentrations do not, however, reflect the rate of turnover of the particular substance. In all probability, the local release of different chemical agents partially explains the different response of the circulatory system. It is indeed true that these local substances may be carried into the systemic circulation if produced in excess.

The Catecholamines

The catecholamines are known to participate in the genesis of shock and the body's response to shock. The initial effects of these substances are related to the ionotropic and chronotropic stimulation of the heart, constriction of the arterioles in the skin, kidney and viscera, dilatation of arteries in striated muscles and myocardium and finally constriction of most veins. These effects help maintain circulation to the brain and heart but there is strong suggestive evidence that the increase in circulating catecholamines may contribute to the magnitude of tissue ischemia which is characteristic of most forms of shock. The metabolic effects are related to increased glycogenolysis and hyperglycemia.

It is thought that the catechols may act upon the energy — poised systems within the cell and effect a disturbance in the ionic distribution which would lead to changes in the contractile elements in the cells. Thus local shifts in the redox potential may be an important factor in the effectiveness of the catechols. Also catecholamines and other hormones may produce electrolyte and pH shift at the cell membrane early in the course of their metabolic action.

Histamine

Widely distributed throughout all tissues, histamine is associated with the granules of mast cells where it is in loose coordination with heparin. The mast cells are usually situated close to blood vessels, therefore histamine can be rapidly disseminated throughout the body.

Histamine is a vasodilator substance and can produce increased tissue permeability. Schayer[4] has suggested that a balance between histamine and catecholamine exerts the main control over circulatory homeostasis and that histamine is the main regulator of the microcirculation. This was based on the observation that there is an increased synthesis of histidine decarboxylase in shock tissues. This may imply that the local synthesis of histamine rather than preformed histamine in mast cells is the important physiologic mechanism controlling the microcirculation. The increased histamine synthesis may produce small-vessel damage which may result in increased capillary permeability. There is also another indication[11] that the increased permeability results from post-capillary venular spasm rather than from a direct toxic effect on the endothelial cells. To sum up, systemic histamine in large doses can produce severe hypotension.

Serotonin

This is a vasoactive substance present throughout the body but has been found in largest concentrations in the brain, G.I. tract, platelets and mast cells. It has been shown to be released in both experimental and clinical shock and during prolonged pump oxygenation when there is continued trauma to the solid elements of the blood. Its action on the vascular musculature is variable depending on the condition. It can raise or lower the blood pressure and its effects on vessel tone are usually opposite to existing physiochemical conditions.

Polypeptides

Bradykinin and Kallidin II are potent vasodilators produced by the action of enzymes such as trypsin or kallikrein on bradykininogen and kallidinogen respectively. The levels of these two active substances have been observed to increase in both clinical and experimental shock. The polypeptides, particularly bradykinin, produce dilatation of vessels of the skin and muscle in amounts comparable to histamine. Bradykinin potentiates the localized Schwartzman and Thomas reactions.

Angiotensin II is also vasoactive and is produced by the action of renin on a plasma substrate, angiotensinogen. Its action is opposite that of bradykinin and kallidin. It causes an increase in the total peripheral resistance, central venous pressure and arterial pressure with a decrease in cardiac output.

Metabolites

With tissue hypoxia, diminished venous blood flow and impaired lymph flow, various metabolites accumulate. These metabolites may affect local tissue circulation and metabolism as well as circulation of other areas to which they are eventually carried. Initially, the local metabolites relax the pre-capillary sphincters more than the post-capillary sphincters. If the shock condition continues, the resistance of the post-capillary sphincters exceeds that of the pre-capillary sphincters. These effects are consistent with the development of the oedema of shock.

From these considerations, the microcirculation is an essential area of concern in shock. Thus, the prevention of capillary stagnation is an urgent necessity to reduce the concentration of metabolites and lessen the possibility of intravascular coagulation or blockage induced by viscosity and aggregation tendencies.

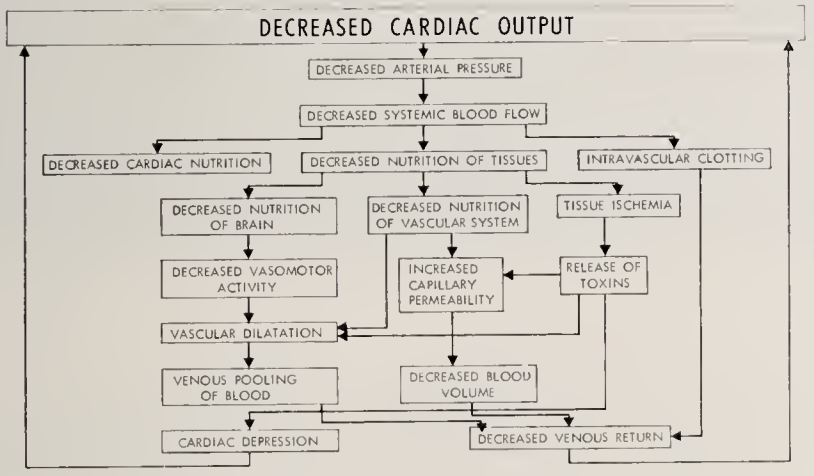
CHANGES IN KEY ORGANS

Heart (the pump):

The role of the heart in shock has not been thoroughly investigated; this may be due to the fact that cardiac deterioration is often masked by the tremendous cardiac reserve. The heart can deteriorate to less than one fifth

and sometimes one third its normal pumping strength without any measurable evidence of cardiac failure. It has been maintained by many investigators that the deterioration of the heart is one important cause of progression and irreversibility in shock[5]. Others hold the contrary view that decompensation of the peripheral circulation is the essential feature of clinical shock. This view was based on the observation that the heart is able to function quite competently with good output if it is provided with adequate venous return and reduced peripheral resistance[6]. Obviously, it seems logical to think that there are instances when either the heart or the peripheral circulation may be the dominant factor. The subjective question probably is not which factor is worse or which factor deals the final blow, but rather to what extent is each factor operative in each specific case.

In shock, the myocardial cells are inadequately perfused despite coronary artery autoregulation. Cardiac contractility is further hindered by the metabolic acidaemia and the circulating substances arising from injured cells elsewhere. The resulting inadequate pumping sparks off the vicious cycle, shown in fig. 1, when there is a significant fall in cardiac output. Progressive shock results.



The Vicious Cycle in Shock[5]

The Lung

Although pulmonary alterations are not the earliest events in shock, they frequently play a major role in late complications. In hemorrhagic shock for example, there is a progressive diminution in the elasticity of the lungs with resultant pulmonary congestion and edema. Hypercarpia and hypoxia result from these changes.

The histological changes reflect clogging of the pulmonary capillaries with red cells, and edema of the interstitial tissues. Patent alveoli are filled with cellular debris and edema fluid.

These pulmonary manifestations are due to 1) changes reflecting left-sided heart failure due to over-zealous administration of fluid to a failing heart 2) changes that are part of a sympathomimetic response and some humoral agents. Histamine, epinephrine and nor-epinephrine increase the pulmonary venous resistance which tends to limit the volume of blood transfusion and plasma expanders that may safely be administered. Excess intravenous fluid gives rise to pulmonary edema and decreased oxygen exchange, loss of surfactants and a higher incidence of pulmonary infection and possibly atelectasis. When transfusion therapy adversely affects the lungs it is probably because the pulmonary circulation is the first capillary network reached by the intravenously administered blood.

The Kidney

Early after the onset of moderate and severe degrees of shock, renal blood flow and function (glomerular filtration and tubular reabsorption) are decreased. Renal ischemia is an unquestionable factor in the genesis of acute renal failure. The ischemia is the result of prolonged vasoconstriction of the kidney in response to hypotension. During experimental hemorrhagic shock to arterial blood pressure of 40 - 60 mm Hg, renal blood flow may fall from 25% to less than 7% of the cardiac output while renal vascular resistance may increase approximately sevenfold[7].

The effects of the prolonged renal vasoconstriction are increased interstitial pressure producing the swollen pale "shock kidney" and focal tubular necrosis with oliguria. The anatomical changes are not as pronounced as the functional derangements. The cortical areas are more severely affected than the medulla. This is because, in normal circumstances, circulation is rapid in the cortical area and the oxygen tension is high, while in the medulla the circulation is slow and the blood is relatively desaturated.

Changes in renal function may vary from simple oliguria with concentrated urine which is acidic, to high output renal failure with low specific gravity and high pH of the urine or to pure renal failure. Renal insufficiency without oliguria is important from the standpoint of recognition and clinical management of a variant of the classic acute renal failure. The chief danger of high output renal failure are 1) failure to recognise the existence of renal failure because of normal output and 2) the administration of potassium salts intravenously. Clinical experience and laboratory experiments suggest that high output failure represents the renal response to a less severe or modified episode of renal injury than that required to produce classic oliguric renal failure.

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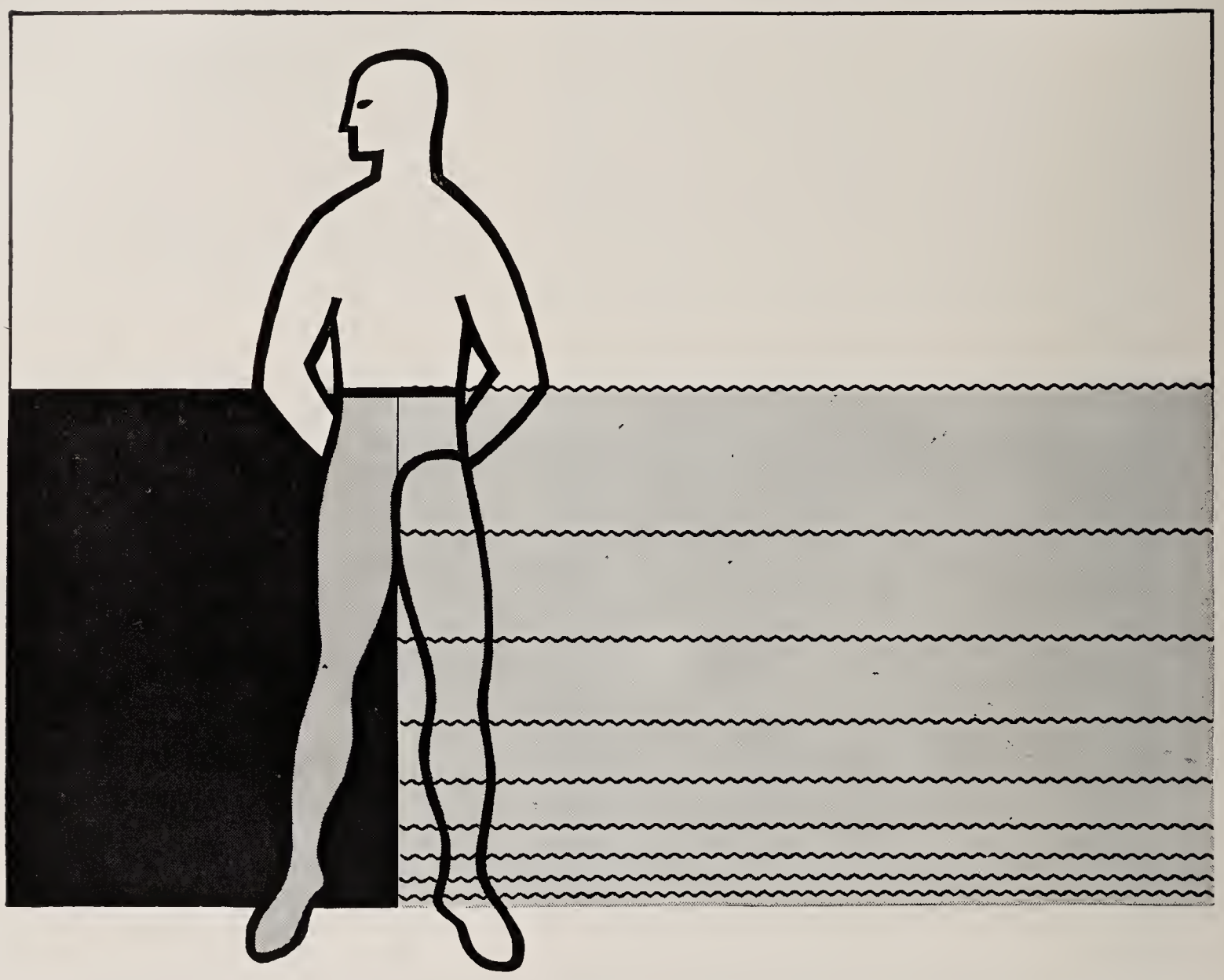

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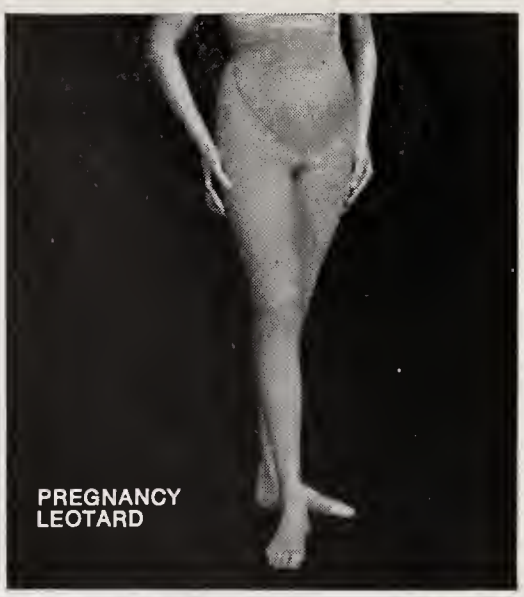

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nitrogen that may require therapy. Changes in ion concentration, including a rise of serum potassium, are dependent on adrenal cortical response, the change in metabolism from aerobic to anaerobic with resultant release of K+ and specifically on changes within tissues involved by the shock. If renal function is impaired the concentration of K+, Mg+ and creatine can rise to be quite high in serum. The rise can be short-lived if renal function is maintained. Potassium will be excreted during the recovery phase of shock.

Although urine flow may return after variable periods of oliguria, the damage to the kidney may persist for several months.

Liver

The initial response of the liver to shock is an increase in its blood flow and this may lead to increased hepatic vascular resistance which, if prolonged, may affect hepatic metabolism and contribute to sequestration of blood in the splanchnic area. If blood volume is already diminished, the hold-up of blood in the hepatic and splanchnic circulations may be tolerated poorly. Delay in the return of venous blood from the splanchnic area may also limit cardiac output and compromise effective circulation, even if the total blood volume is normal.

Changes at the cellular level include enlargement and swelling of the mitochondria. There is an increase in the oxygen consumption of the cells, indicating that the liver is in a state of over-activity.

The engorgement of the liver with associated increased vascular resistance is present in a wide variety of conditions leading to shock and it may be a common underlying factor in the development of the shock syndrome.

THE HOMEOSTATIC MECHANISM IN SHOCK

In addition to considering important changes in the body during shock, a brief discussion of the interaction of the initiating factors and the complex body responses to stress will provide a reasonable understanding of the development of the shock syndrome. Hemorrhage, the commonest of the various causes of traumatic shock will be described to exemplify the complex body response.

The primary effect of hemorrhage is hypovolemia. Initially, impulses are sent to the medullary centre controlling the cardiovascular system by the stretch receptors, especially the carotid and the aortic bodies. The result is an increased autonomic nervous system activity. The blood stream is immediately flooded with catecholamines, most probably produced by the adrenal medulla. There is a concomitant increase in the heart rate.

The secondary reactions of the body to blood volume deficit is an increased production of antidiuretic hormone and aldosterone. Renin and angiotensin II are produced in increasing amounts. An increase in the production of glucocorticoids is currently disputed.

The effects of the initial response on the circulation is an increased pulse with vasoconstriction. There is an initial increase in cardiac output followed by a decrease in cardiac output as explained earlier. Because of these effects on the circulation, there is an altered distribution of cardiac output between vital organs like the heart, brain and the muscle mass. Sodium and water are conserved with decreased urinary output. Additional fluid intake is encouraged by the profound thirst that develops. Respiratory response to oligemia alone is one of an increase in rate and depth, proably related to the baroreceptors. However, acidosis appears very promptly after the development of oligemia and is undoubtedly a potentiating factor.

The effects of the circulatory changes are decreased tissue perfusion, decreased hepatic and renal functions and depressed central nervous system activity. If homeostassis is not restored, the injury becomes overwhelming and self-perpetuating. The perimeter defenses are abandoned and all effective strength is concentrated on the vital areas for a last stand. If shock still persists, tissue metabolism in all organs in the body is further decreased. Treatment can be of little help at this stage and death eventually results.

Septic and antigen-antibody reaction shock conditions are variants of the hemorrhagic shock described above. These two conditions have been adequately reviewed. The reader is referred to [8 9 10] for further details.

Shock in man is usually complex because the initiating factors are often multiple. The patient's general physical status and medical background are unique and the physiologic responses to similar noxious events are not constant. Therefore, an understanding of the pathophysiologic mechanism of shock is essential for a critical analysis of the signs and symptoms. The following management procedures may be of some help in instituting a rational therapy of the shock syndrome:—

1) A continuous monitoring of the systemic arterial pressure.
2) Monitoring of the central venous pressure.
3) Monitoring of urine flow.
4) Monitoring of the blood volume.
5) A thorough laboratory investigation of the blood chemistry, the blood gases, pH and electrolytes.

And finally, a constant bed-side attendance is necessary so that any physiologic nuances that may occur can be rectified with appropriate therapeutic adjustments.

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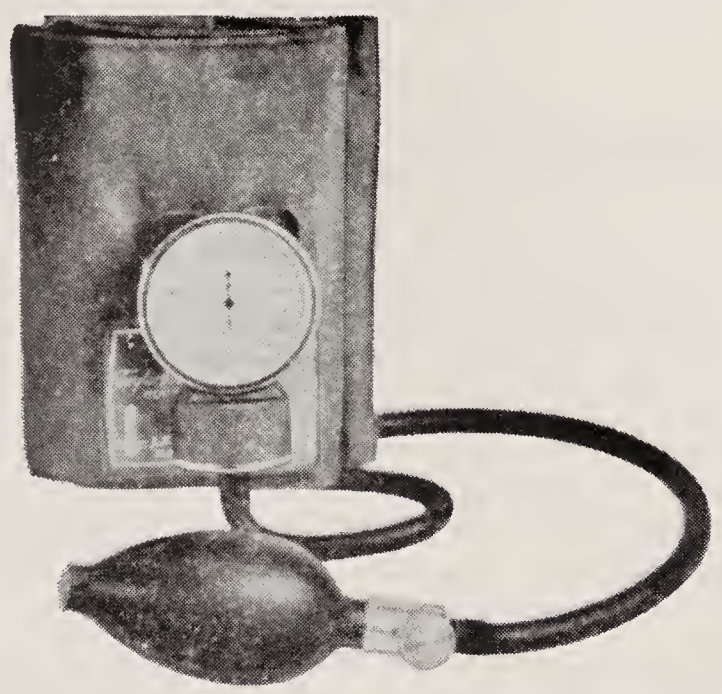

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A STUDY OF TRAUMA AT THE CELLULAR LEVEL

PHYSICAL AND BIOLOGICAL PROPERTIES OF MICROWAVES

The microwave band of the electromagnetic spectrum ranges in wavelength from less than a centimeter to about 30 meters. This wavelength is longer than infra red, visible and ultraviolet light, X-Rays and gamma rays, but is shorter than radio or short waves. The frequency of microwaves ranges from 10^7 to 3.0 x 10^{11} cycles per second. Microwave electromagnetic nonionizing (M.W.) radiation provides a source of heat energy which can be readily controlled and can be delivered to a specimen without actual physical contact.

When a substance is exposed to microwaves, polar molecules align themselves with the electromagnetic field. Because this field is rapidly changing, these molecules vibrate, producing heat. Energy is thus delivered to specimens 100 - 1,000 times faster than with other heat sources.

A further advantage of M.W. radiation is that heat is delivered uniformly throughout a substance without surface localization.

M.W. radiation has been used in a wide variety of biological systems. Exposure of tissues has been shown to cause a heat-induced inflammatory reaction. Following this initial response, subsequent tissue development proved to be beneficial in certain circumstances and detrimental in others. Exposure of tissues has been shown to: a) cause leukocytosis and eosinopenia in dogs[1], b) inhibit experimental arthritis in rats[2], c) reduce the growth of liver hepatomas in rats[3], and d) inhibit the growth of kidney tubular cells in rats.[4] In addition, microwaves have been used in the treatment of iritis, scleritis, malignant melanoma of the choroid, cyclitis, post-operative thrombosis, and muscular degeneration.[5,8]

Microwave exposure causes a direct thermal effect and an indirect non-thermal reaction. The precise macromolecular target of the thermal effect is uncertain. This site of action could be at the level of specific protein molecules, through the vibration of polar molecules in their surrounding solution. Supporting this theory is the observation that large protein molecules (eg. — gamma globulin) have been cleaved by M.W. exposure.

The non-thermal effect of M.W. exposure is partially attributed to the production of ionized gas plasmas, which are able to catalyze chemical reactions. Free electrons present in a gas such as hydrogen can be accelerated by microwaves to higher energy states, causing ionization. When a sufficient number of electrons reach this energy level, the gas breaks down and forms a plasma, which can facilitate a variety of chemical reactions.

Numerous other non-thermal effects of M.W. exposure have been suggested[5], but few have been convincingly demonstrated.

METHOD OF STUDY

To further elucidate the interaction between M.W. radiation and complex biological systems, studies have been initiated at the cellular and molecular levels[6]. In an attempt to separate the thermal from the non-thermal effects of radiation, cells have been treated with a prototype M.W. distributing unit (Gray Industries, Fort Lauderdale, Fla., patent pending) which has a temperature regulating system. Various cell lines have been employed including: normal human epithelial cells, epithelial cells obtained from a patient with Morquio's disease (a form of osteochondrosis), human leukocytes, L-cells, (a standard strain of mouse fibroblast) epithelial cells isolated from a rabbit carcinoma, and mesenchymal cells isolated from a rat osteogenic sarcoma.

Cells were treated with single and multiple pulsed exposures, and their subsequent biochemical properties were compared to those of untreated cells. Measurements were made of the rate of cell growth, uptake of radioactive substrates, and distribution and intracellular stability of these substrates. Biochemical properties were correlated with morphological changes, as determined by routine histology, histochemistry and electron microscopy.

RESULTS

Preliminary experiments indicated that certain cell lines are more susceptible to M.W. radiation than others. The rate of cell growth and the overall metabolic rate of these sensitive cells was markedly decreased following treatment. These growth aberrations were manifested 24 to 48 hours after treatment and persisted for 10 days, when the cultures became saturated. One particular line of tumour cells appeared to be highly sensitive to exposure.

The delay in appearance of altered metabolic activities suggested that the effects were not expressed until the cells completed their duplication cycle. The persistance of the observed growth abnormalities through several cell cycles is difficult to explain. An attractive hypothesis is that M.W. exposure has produced an acti-

vator which influenced the level or activity of specific enzymes. Further experiments are in progress to determine the nature of these observations.

Studies are also being conducted using tumour-bearing animals. Prior to exposure, tumours have been injected with metabolically inert materials which serve to localize the thermal effect of M.W. radiation. Application of microwaves to tumours *in vivo* could be particularly advantageous because the heat induced inflammation reaction impairs tumour blood flow and produces anoxia.[3] This local condition could serve to reduce or inhibit subsequent tumour growth.

In addition, many organs which are resistant to other forms of radiation are extremely sensitive to M.W. radiation.[5]

Over the past years considerable knowledge has been accumulated concerning the mechanism of action of various types of radiation, including ultraviolet light, gamma rays and X-Rays. Relatively little is known however, of the specific effects of M.W. radiation. Further experiments, using specific cell types and tumour-bearing animals, treated with a rigidly controlled source of radiation in a temperature regulated environment, may lead to a clearer understanding of the mode of action of M.W. radiation.

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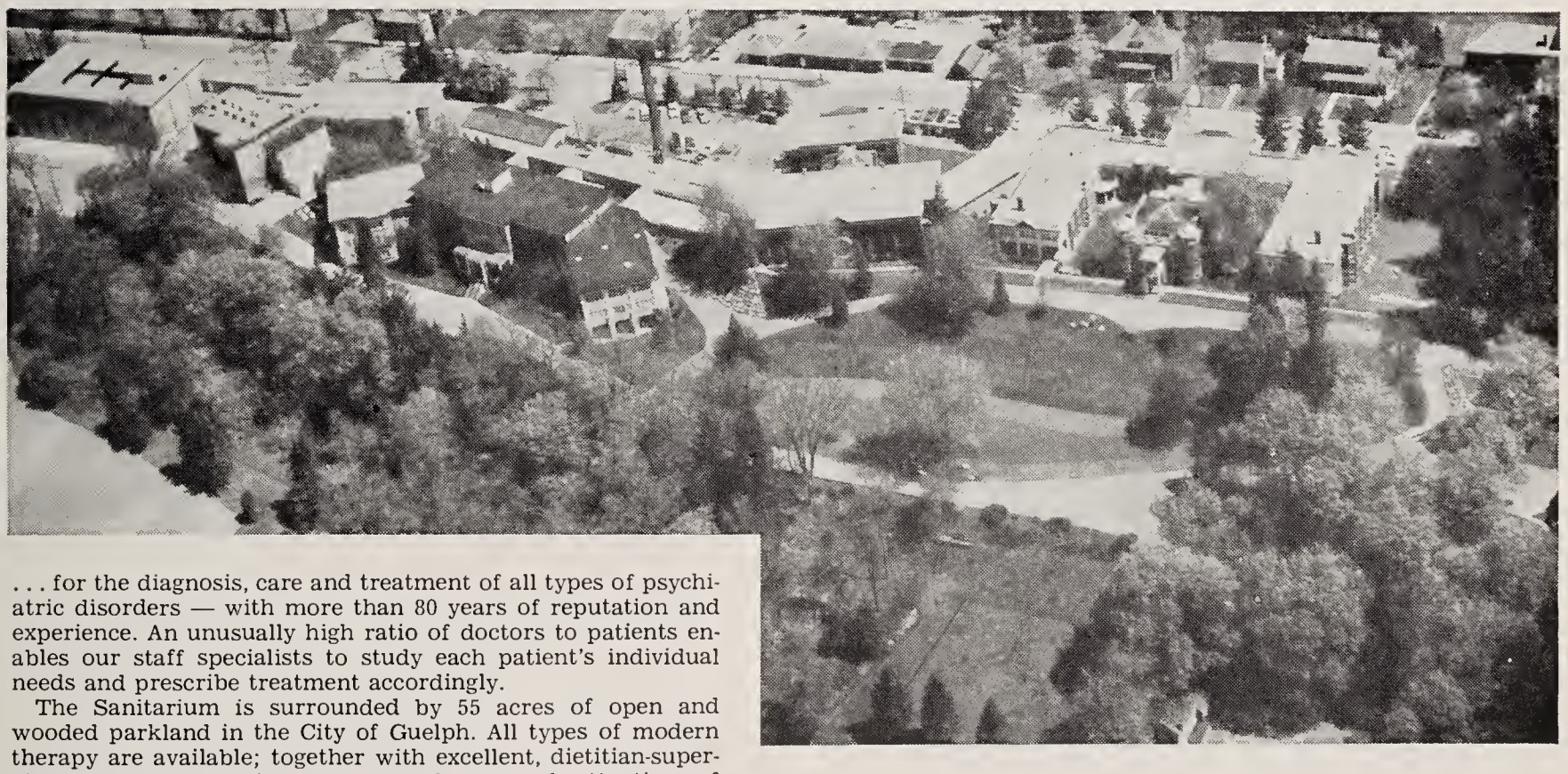

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Back Trauma

R. W. Ziedenberg 7T0

Phylogenetically, the spinal cord was enveloped in a bony vertebral case for support and protection against external penetrating forces. But today, man's changing patterns of civilian living have exposed the back to the hazards of closed injury caused by ever increasing accidents — notably automobile accidents and falls.[7] It is this relationship between the spinal cord and vertebral column which creates a precarious situation wherein the cord is vulnerable to damage in its bony confinement.

This paper will deal with vertebral column and spinal cord injuries under the following headings:

(a) FRACTURES & DISPLACEMENTS OF THE SPINE

(b) NEUROLOGICAL LESIONS AND COMPLICATIONS OF FRACTURES OF THE SPINE

(c) MANAGEMENT OF THE ACUTE SPINAL INJURY

(A) FRACTURES AND DISPLACEMENTS OF THE SPINE

Classification [1 2 3]

A study of the types of injuries incurred by the spine requires an understanding of basic anatomical structures. Articulation of the vertebrae (fig. 1) is accomplished by the synarthroses between the vertebral bodies and the two posterior diarthrodial apophyseal joints, both components being held together by a series of ligaments. While the strong annulus fibrosus, the anterior longitudinal ligament, and the posterior longitudinal ligament stabilize the vertebral bodies, it is the *"posterior ligamentous complex"* which supports and binds the posterior apophyseal joints. The complex is composed of the capsules of the posterior joints, the ligamentum flavum, the intraspinous ligaments, and the supraspinous ligaments. Furthermore, the posterior ligamentous complex is the key to the classification of the major spinal injuries and determines their stability.

The type of fracture, dislocation, and fracture-dislocation depends on: (1) the type of violence (flexion, flexion and rotation, extension, and compression)[4]; (2) the integrity of the posterior ligamentous complex, that is, whether or not it is ruptured; and (3) the region of the spine involved (because of the shape of the bones and the articular processes).

In general, violence resulting in pure hyperflexion or violence transmitted along the line of the vertebral bodies rarely, if ever, tears the posterior complex, while flexion-rotation almost always does. It is the rotational element which determines the liability to rupture. If the complex is intact, then the fracture is stable, and further displacement of the fragments will not occur, unless there is additional severe trauma. If it is ruptured, the resulting fracture, dislocation, or fracture-dislocation is unstable and the fragments can be displaced easily by minimal forces.

Pure Flexion Injuries

With the posterior complex intact and the posterolateral joints not dislocated, the vertebral body is crushed, resulting in a *simple wedge fracture* (fig. 2) as the flexion force is expended. Because the stability of the thoracic spine is increased by the rib cage, and the posterior ligament complex in the lumbar spine is extremely strong, this wedge compression fracture is commonest in the thoracic and lumbar spines. Stability of the spine is ensured by the impaction of the fragments, the integrity of the posterior complex, and the proper alignment of the apophyseal joints.

Flexion-Rotation Injuries

When the posterior ligament complex ruptures, the posterior tie is lost, and if the amount of flexion is sufficient to disengage the articular processes, then *pure dislocation* results. Because there is no fulcrum for lever, no vertebral body compression occurs. A pure dislocation is common in the cervical spine where the amount of flexion to disengage the articular processes is slight, rare in the lumbar region where a great amount of force is necessary, and even more unlikely in the thoracic area where the supporting rib cage is present (fig. 3). These injuries are unstable.

When this type of violence occurs in the lumbar spine, the posterior ligaments rupture, one and/or the other articular process fractures, and the upper vertebra swings upon the lower, carrying with it a wedge slice of the upper border of the lower vertebral body. (fig. 3). This very unstable injury, often associated with paraplegia, gives rise to the classical *rotational fracture-dislocation* of the thoracolumbar and lumbar spines (fig. 4).

Extension Injuries[5]

In this injury (commonly experienced in "whiplash" of the neck), the anterior longitudinal ligament frequently ruptures with a resultant extension subluxation (fig. 5). The spinal cord may or may not escape injury. There is stability while the neck is in the neutral position or in flexion.

Compression Injuries

When the cervical (and rarely the lumbar) spine is held straight, vertical forces are transmitted directly along the line of the vertebral bodies, one of the end plates ruptures, and the nucleus of the disc is driven

into the vertebral body which explodes. This trauma produces the compression *"burst" fracture* which is associated with tetraplegia in the cervical region when the cord is damaged by posterior fragments blown peripherally (fig. 6). All ligaments remain intact and the spine is stable. This compression force may also fracture the anterior and posterior arches of the atlas, causing lateral displacement of each half of the atlas. This not uncommon injury is termed Jefferson's fracture.

Diagnosis

Early diagnosis of this type of injury is crucial because the treatment and prognosis depends upon an accurate assessment of the damage to the spine.

On inspection, abrasions and contusions over one scapula or on one side indicate a rotational element at the time of a flexion injury at the thoracolumbar level. This is in contrast to the central bruising, occurring in pure flexion. Employing palpation, one can detect gaps between affected vertebrae due to the rupture of the supraspinous and intraspinous ligaments, and it is this physical sign which is the best indicator of spinal instability. Radiological confirmation is useful because each type of fracture and fracture-dislocation has a characteristic appearance. It must be remembered, though, that not all injuries will be accompanied by demonstrable radiographic displacement.

Treatment

Restoration of a painless, powerful, mobile back is best achieved by obtaining stability at the affected segments, regardless of anatomical mal-alignment.

If the vertebral body or posterolateral articular processes have been fractured, spontaneous inter-body fusion almost always occurs during healing (except with wedge fractures), and complete stability is ensured independent of ligamentous healing. On the other hand, in pure dislocation, fusion does not always occur because there is no fracture and reconstitution of the posterior ligaments does not provide reliable or permanent stability.[6 1 2]

Some of the guiding principles are summarized in Table 1. Treatment of vertebral injuries with paraplegia and tetraplegia requires modifications in order to preserve the remaining neurological function and to prevent further deficits.

Thus it becomes evident that the weakest points in the spine — the points of junction of normally directed curves: anteriorly convex in the cervical region, posteriorly convex in the thoracic, anteriorly convex in the lumbar, and posteriorly convex in the sacral — bear the brunt of the stresses applied in horizontal and vertical planes. It is no wonder, then, that force in the vertebral plane results in damage to the following areas: vertebrae C5 to C7, T12 to L1, and L4 and L5 to S1.[7] Furthermore, the injuries sustained depend on: (1) the shape, size, and orientation of the vertebrae and their articular processes, (2) the articulating and stabilizing structures, and (3) the characteristics of the traumatic forces applied.

(B) NEUROLOGICAL LESIONS AND COMPLICATIONS OF FRACTURES OF THE SPINE

The most serious complication of vertebral column injury is spinal cord and nerve root damage. In most cases the skeletal lesion is in the thoracic spine or at the thoracolumbar junction; less frequently in the cervical or lumbar region.[6]

Injury to the spinal cord and nerve roots results in paralysis of the bladder and bowel and anaesthesia below the damaged level. This may be complete or incomplete.[3]

Lesions of the Cord

Complete transection of the cord results initially in total suppression of function in the segments below the lesion with flaccid paralysis, complete sensory loss, and absent visceral reflexes during the phase of *"spinal shock"* which lasts up to a few weeks. Gradually, this period passes and the resultant state is characterized by spastic paralysis, and exaggerated tendon and visceral reflexes isolated from cerebral cortical modification. Because the cord has no regenerative powers, these changes are permanent.

If the lesion is incomplete, the only indication of continuity of axons in the spinal cord is the preservation or early return of voluntary motor power and sensation below the level of involvement.

Lesions of the Nerve Roots

It is with some optimism that one hopes for recovery with cauda equina damage because the motor nerve root behaves like a peripheral nerve, and if the fibrous epineurium of the fasciculi remains intact, regeneration is possible. This does not apply to the sensory components of the cauda equina because the sensory ganglion cells lie outside of the spinal canal and if the lesion is located between the cord and the ganglion cells, recovery will not take place.[8]

Because of the possibility of recovery, the distinction between paralysis and anaesthesia due to spinal cord damage from that due to root trauma cannot be overemphasized.

The Bladder in Cord and Cauda Equina Injuries

Normally, the cerebral cortex overrides the reflex centres in the second and third sacral segments responsible for bladder emptying.

With a transection of the cord above the sacral segments cortical control is abolished and urinary retention results initially until the reflex centres become in-

Table I: FRACTURES AND DISPLACEMENTS OF THE SPINE AND THEIR TREATMENT

Type of Injury	*Force*	*Region*	*Stability*	*Treatment*
Simple Wedge Fracture	Flexion	Thoracic Lumbar (Cervical)	Stable	Bed rest (2-3 wk.) followed by active mobilization for 6-8 wk.
Pure Dislocation	Flexion-Rotation	Cervical	Unstable	With or without neurological deficit traction, reduction and fusion.
Fracture-Dislocation	Flexion-Rotation	Cervical Thoraco-lumbar Lumbar	Very Unstable	Without neurological deficit, reduction and immobilization (approx. 4-5 mo.). With neurological deficit, but where there is hope of recovery, reduction and fixation (3 mo.)
Extension Dislocation Burst	Extension	Cervical	Stable in Flexion	Stabilize in neutral position for 2-3 mo.
Fracture	Compression	Cervical (Lumbar)	Stable	Immobilization for pain for 3-4 mo.

Table II: VERTEBRAL INJURIES AND NEUROLOGICAL DYSFUNCTION

Region	*Injury*	*Neurological Deficit*
Cervical	Dislocation Fracture-Dislocation Burst Fracture	Complete or incomplete cord injury
Thoracic	Fracture-Dislocation	Nearly always complete cord transection
Thoraco-Lumbar	Fracture-Dislocation	Mixed cord and nerve root lesions
Lumbar	Fracture-Dislocation	Nerve roots (cauda equina) complete or incomplete division

dependent after one to three months. The result is an *"automatic" or "cord" bladder,* which empties automatically when a certain capacity is reached.

When the sacral segments of the cord or the nerve roots emerging from it are cut, the reflex control is lost. Periodic emptying of this *"autonomous" bladder* depends on local reflexes in the bladder wall itself which can be aroused by manual compression of the abdominal wall. The emptying of this bladder is less complete than it is with the automatic bladder.

Total Paraplegia: Permanent Damage Vs. Spinal Shock

Another important concept to understand entails the difference between total paraplegia due to irreparable cord damage and that due to a partial lesion associated with spinal shock. Both can present with complete paralysis and anaesthesia below the level of the lesion. This is a puzzling situation which may lead to confusion in treatment. If the cord damage is permanent, there is no hope of recovery. If spinal shock is present, immediate management of the vertebral and spinal cord injuries can be of the utmost importance.

It is usually stated that spinal shock may persist for up to three or more weeks, and thus, it is impossible to be certain whether the cord is irreparably damaged for several weeks! This is false. Holdsworth found that in every patient where the initial physical examination showed complete paraplegia, unless some recovery of motor power or of sensation occurred in the first 24 hours after the accident, no subsequent recovery took place.[3] However, the return of the slightest movement or sensation pointed to a better prognosis.

To separate complete from partial transection requires a meticulous neurological examination of all modalities of sensation in every dermatome, each muscle, and all reflexes every few hours. If any power or sensation is present, continuity of the long tracts is ensured, but, the presence of any reflexes with the total absence of power or sensation indicates the isolation by

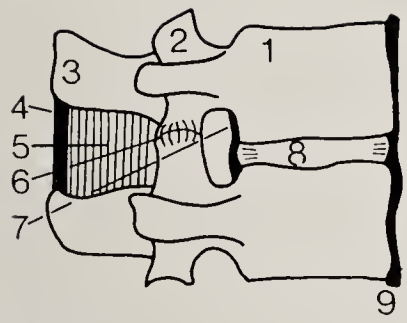
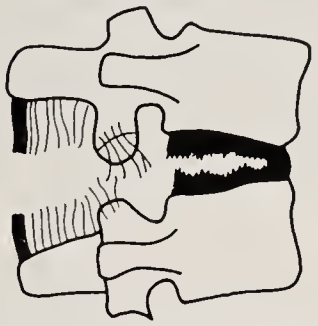


Figure 1
Articulation of the Vertebrae

(1) Vertebral Body
(2) Superior Articular Process
(3) Spinous Process
(4) Supraspinous Ligament
(5) Intraspinous Ligament
(6) Posterior Joint and Capsule
(7) Posterior Longitudinal Ligament
(8) Intervertebral Disc
(9) Anterior Long. Ligament

Figure 3
(a) Cervical Spine Dislocation with Forward Displacement



(b) Lumbar Spine Dislocation. More Flexion is Needed.

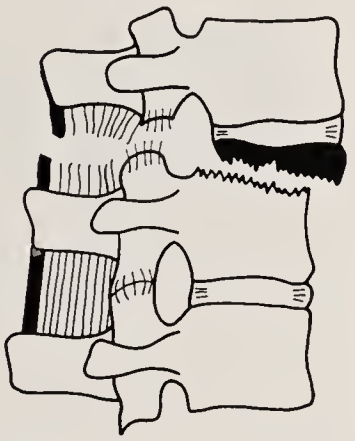


Figure 4
Fracture-Dislocation of Lumbar Spine with Rotation

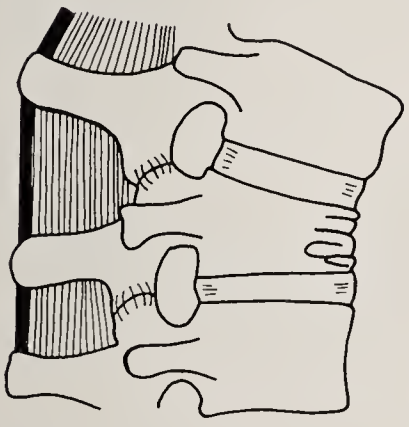


Figure 2
Wedge Compression Fracture with an Intact Posterior Complex

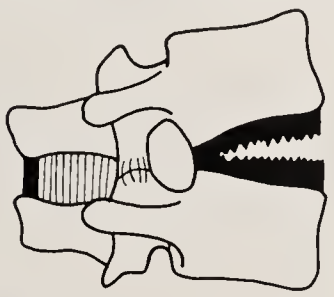


Figure 5
Extension Injury with Intact Posterior Ligaments and Ruptured Ant. Long. Ligament ("Whiplash" of the Neck)

division of the long tracts of that section of the cord responsible for the reflexes — a bad sign indeed!

The Distinction Between Cord and Root Damage

If there is no recovery within 24 hours following a cord injury, no subsequent improvement can be expected. This does not apply to paralysis with root lesions. They may take months to regenerate, but they often do.

Initially, there is no difference between the defects of cord and root damage on examination, but a study of the clinical and radiological findings combined with a knowledge of the anatomy of the cord, roots, and vertebral column can unravel the mystery (fig. 7).

In the cervical region the neurological segment is almost opposite the corresponding vertebra, but in the lower thoracic and upped lumbar spine there is marked disparity, and the spinal nerve roots travel increasingly oblique courses. This is exemplified by the fact that all lumbar and sacral cord segments lie between vertebra T9 and the lower border of vertebra L1 (where the cord ends). Opposite disc space T12 - L1 lies the first sacral segment of the cord and all the lumbar nerve roots.

If we assume that the nervous tissue damage is at the level of the fracture or dislocation, the anatomical site of damage can be ascertained by x-ray. A clinical neurological examination will determine the upper level of the deficit. The comparison of the radiological with clinical findings will then reveal how much paralysis and anaesthesia is due to cord damage and how much is root.

Therefore, injury to the cervical vertebrae results in almost pure cord lesions because of the juxtaposition of vertebral and cord levels. Below the first lumbar vertebra, only pure root lesions are possible because the lumbar region of the canal contains only nerve roots. With fracture-dislocations at the thoracolumbar junction, mixed cord and root lesions occur (fig. 8). The characteristic injury for specific spinal regions is summarized in Table II.

Treatment of Vertebral Injuries with Neurological Deficits

In the presence of paraplegia or tetraplegia: (1) reduce the deformity, (2) restore alignment of the spine and the normal anatomy of the vertebral canal, and (3) stabilize the unstable injuries.

Because skull traction does not interfere with nursing, cervical spinal injuries are treated the same with or without nerve injury. In the thoracic spine direct violence usually inflicts severe displacement and complete transection of the cord. With complete paraplegia, management of the vertebral damage is less critical. The thoracolumbar and lumbar fracture-dislocations often give rise to incomplete nerve injury, thus necessitating open reduction and internal fixation.

(C) MANAGEMENT OF THE ACUTE SPINAL INJURY

In caring for the accident victim, one must strive for two ultimate goals: (1) the preservation of life and (2) the curtailment of disability.

The most serious situation encountered in spinal trauma involves unstable damage to the cervical spinal

cord, as exemplified by: fractures of the base of the odontoid process causing atlanto-axial fracture-dislocations, hyperextension violence resulting in momentary soft tissue displacement (the nucleus pulposus of a disc or a fold of ligamentum flavum), or flexion-rotation of the lower half of the cervical spine leading to fracture-dislocations, Therefore, when one handles any spinal injury where the level of involvement is in doubt, he should approach it as if it were in the cervical spine. This is the premise for much of the following discussion, since instability is the key-note. Indiscretion can result in tetraplegia or death!

Management at the Scene of Injury[9 10 11 7]

(1) *History* — This can be informative, but the accounts of spectators may be unreliable.

(2) *Interrogation* — If the immediate condition is not critical then briefly ask: (a) where the pain is; (b) whether he can feel the examiner's hand touch him over his arms and legs; and (c) patient to move both hands and both feet.

(3) *Examination of Priorities* — At this time, invoke the CRASH PLAN[9] mnemonic which gives the sequence of priorities:

C — ardio; R — espiratory; A — bdomen; S — pine;

H — ead; P — elvis; L — imbs (lower, then upper);

A — rteries; N — erves (function of the major peripheral nerves).

(4) *Respiration* — Maintain an adequate airway. It is important to consider that the nervous control of both the intercostal muscles and the diaphragm is impaired by injuries to the upper half of the cervical cord, while the diaphragm still performs with lower half damage.

As they are respiratory depressants, narcotics for pain and restlessness are forbidden.

Do not give fluids orally! This avoids unnecessary neck movement and the possibility of vomiting, with the development of aspiration pneumonia.

(5) *Shock — Surgical Vs. Spinal* — Spinal shock may present with hypotension due to a temporary loss of sympathetic vasomotor tone, but it lacks the accompanying tachycardia of "surgical" shock unless the two conditions coexist.

(6) *Transportation* — Without haste, the patient should be placed supine in the neutral anatomical position, the head immobilized between two lateral supports or held by an attendant. In transfering to the ambulance stretcher, four or five persons lift the patient in unison "like a log" with one leader supporting the head and neck while applying slight traction. Canvas traction for temporary neck immobilization during transport is desirable.

The aim of such manoeuvres is to prevent further immediate damage to the spinal cord and nerve roots and to align the spine in order to correct existing neurological defects and prevent future ones.

(7) *Bladder Care* — An indwelling catheter may prevent damage of an overdistended flaccid bladder when the trip to hospital will take several hours.

(8) *Decubitus Ulcers* — Protection of denervated skin from ulceration requires (a) padding of bony pressure prominences (heels, sacrum); (b) smooth, dry sheets; (c) removal of hard objects from the patient's clothing; (d) turning of the patient every two hours in prolonged journeys, and (e) the avoidance of external heat applied to a cold or "shocky" patient.

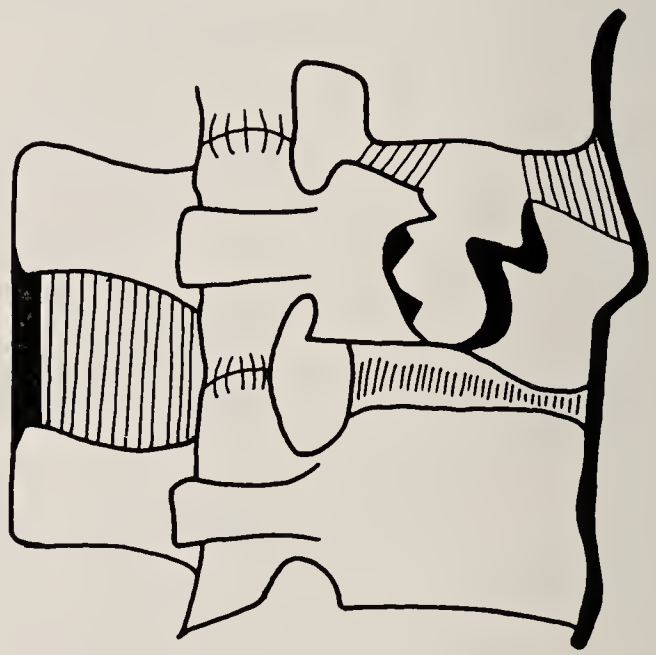


Figure 6
Burst Fracture with Integrity of Post. Ligament Complex.

Management in the Emergency Room

(1) *Vital Signs* — Initially, the level of consciousness, the blood pressure, pulse, respiratory rate, and temperature (rectal) are recorded every 15 minutes to rule out shock. Stabilization of the vital body functions is paramount.

(2) *Associated Injuries* — Search for complications using the CRASH PLAN.

(3) *History* — A brief description from the patient or from witnesses may indicate the mechanism of injury. Whether there was any movement or sensation immediately after the accident and whether there is a change in this status may determine the ultimate outcome. An enquiry into the past health will provide useful information that will influence the general care of the patient.
(4) *Clinical Examination* — After establishing a baseline neurological examination, the same physician should perform the subsequent serial avaluations. The important concepts regarding the neurological lesions have been stated in the previous section of this article. Accord-

ing to Black[2] the level of motor loss (except in the thoracic injuries) is a somewhat more reliable indicator of cord segment involvement than is the sensory level.

(5) *Radiological Examination* — Never move the patient to obtain different views; adjust the x-ray tube, especially in cervical spine injuries. While anteroposterior and lateral views of the affected region are essential, soft tissue damage and easily reducible subluxations may appear normal radiologically.

Special techniques are required in certain cervical injuries. An open-mouth view of the atlanto-axial articulation allows visualization of the odontoid process and possible fractures. Because the lower cervical spine is often obscured by the shoulders, depressing them or burying the film in the muscles of the neck may be beneficial.

(6) *Multiple Spinal Injuries* — The involvement of the column at widely separated levels is rare, but must not be overlooked in patients who are unconscious or in shock.[12] This necessitates a careful clinical and radiological assessment.

(7) *Lumbar Puncture and Queckenstedt Test* — This latter procedure to demonstrate manometric blocks, consists of compressing the jugular veins for ten seconds and observing the fluctuation of cerbospinal fluid in the manometer. The chief value of these manoeuvres in the detection of a spinal fluid block due to cord edema, bony displacement, disc protrusion, or a combination of these factors is doubted by Taylor,[11] who shows that in the presence of lesions higher than C6, the compressed jugular veins are bypassed through the venous vertebral plexuses. This test is contraindicated if intracranial injuries are suspected!

In penetrating wounds, the fluid should be checked for signs of infection.

(8) *Laminectomy* — No longer an emergency measure, it is indicated: (a) in incomplete cord lesions where there are increasing neurological signs suggesting epidural hematoma formation; (b) in injuries in which there is no x-ray evidence of damage, with complete or incomplete paraplegia, or block of spinal fluid; and (c) in depressed fractures of the laminae with protrusion of bone into the spinal canal, especially at the thoracolumbar junction where nerve root rather than cord damage is suspected.[10]

(9) *Skull Traction* — It is indicated in all forms of cervical fractures and/or dislocations regardless of the neurological status. Crutchfield tongs are useful for this job of immobilization.

Management on the Ward

(1) *Nutrition* — The increased protein catabolism early in the course of paraplegia makes the patient susceptible to anemia, urinary tract infection, and the development of pressure sores. Therefore, a 3500-4000

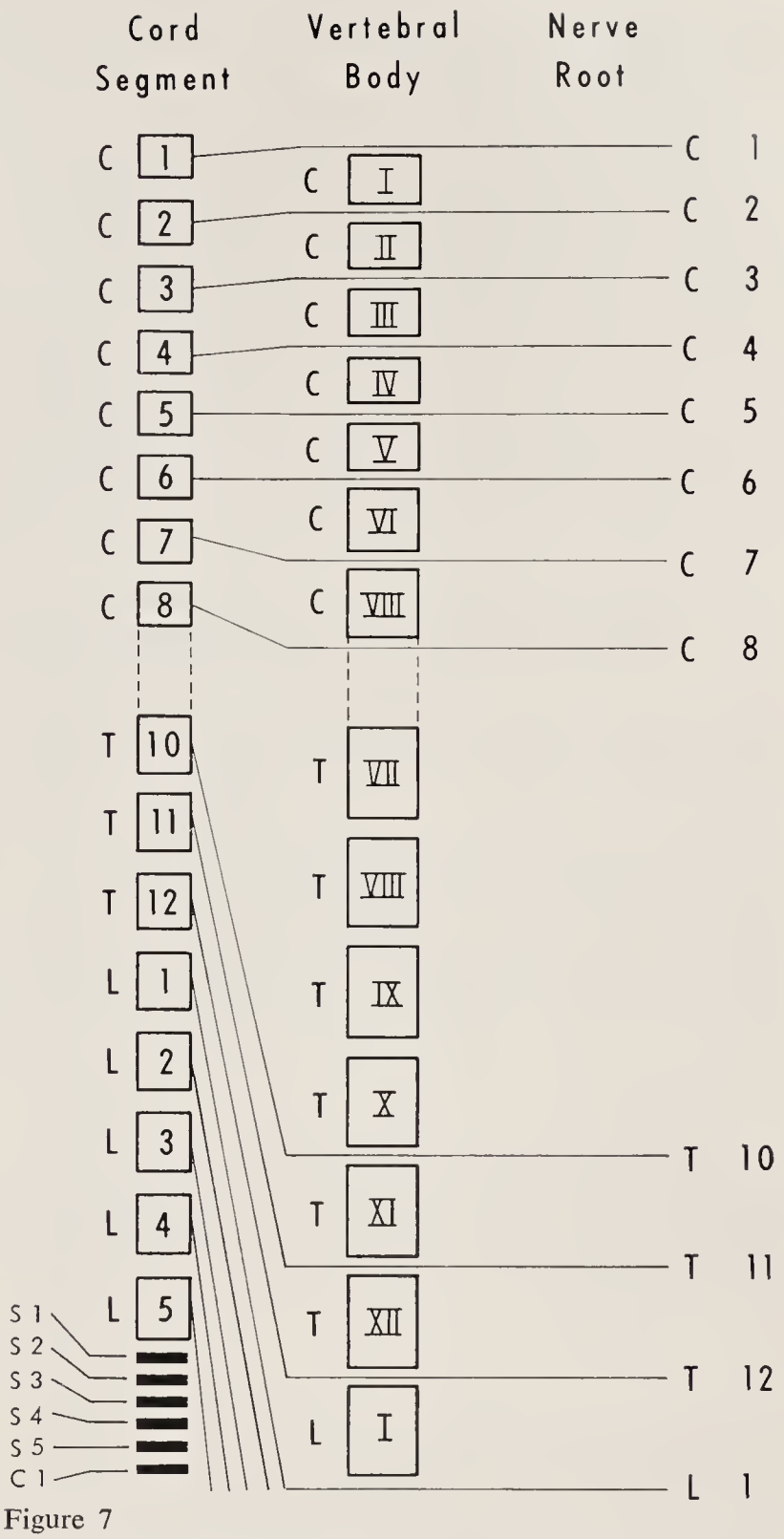


Figure 7
The Anatomical Relationship of Spinal Cord Segments, Vertebrae, and Nerve Roots.

calorie diet with a minimum protein content of 125-150 grams is encouraged daily for an adult.[9 10]

(2) *Skin Care* — The prevention of decubitus ulcers requires the avoidance of prolonged pressure on the skin and the maintenance of a good general condition.

(3) *Neurogenic Bladder* — Because urologic complications — infection and lithiasis — kill at least half the patients with spinal cord injury[11], one must consider the initial treatment of urinary retention. Some physicians prefer intermittent catheterization (three to four times a day to prevent a collection of more than 400-500 ml of urine), while others employ an indwelling Foley catheter with or without tidal drainage. Asepsis is mandatory no matter which method is used.[1 2 4 11]

In bladder training contractures can be prevented by allowing periodic retention.

Although an automatic bladder establishes good function within one to three months, the autonomous or atonic bladder empties less reliably by abdominal straining or by manual compression.

(4) *Neurogenic Bowel* — When treating transient paralytic ileus, one must employ nasogastric suction, rectal tube insertion, and enemas to decompress the bowel. Mineral oil, abdominal massage, glycerine suppositories, digital rectal stimulation, or small enemas should be used to accomplish a movement daily or every other day.

(5) *Physical Therapy* — All paralyzed joints must be put through a full range of passive movements twice daily. Furthermore, daily respiratory exercises are encouraged. A plan of physical therapy aims at preventing contractures and abnormal positions which retard rehabilitation. Some of these manoeuvres are probably prophylactic against deep venous thrombosis of the lower extremities with subsequent pulmonary embolism.
(6) *Blood Chemistry* — Weekly checks will help to detect anemia, negative nitrogen balances, electrolyte losses, acid-base metabolic defects, and other upsets in bodily function.

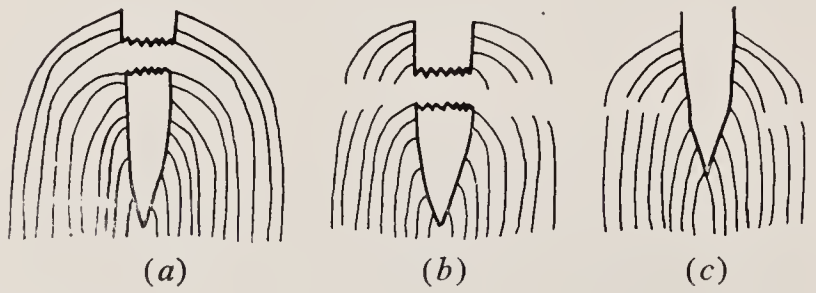


Figure 8

Mixed Spinal Cord and Nerve Root Lesions with Fracture-Dislocations at the Thoracolumbar Junction.

(a) Complete Cord Transection with Root Escape

(b) Complete Cord and Nerve Root Transection

(c) Cord Integrity with Partial Cauda Equina and Root Division

CONCLUSION

In this article on Trauma to the Back I have attempted to emphasize the most important aspect of back trauma — injury to the spinal cord and nerve roots. Consideration of the possibility of such damage must never be forgotten in any case of serious back injury, and the management of a patient in the immediate post traumatic period is basically a matter of preventing further damage to the relatively friable nervous tissue. Improper management at this stage can lead to irreversible paraplegia and even quadriplegia — a very costly and inexcusable error indeed!

ACKNOWLEDGEMENT

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Abdominal Trauma

R. C. Williams 7T0
G. M. Thomas 7T0

INTRODUCTION

There is no doubt today, that with the ever increasing rate of motor vehicle accidents, and of interest and participation in sports, trauma, with particular reference to the abdomen, is of significant interest to each of us as physicians. Auto accidents (68%), falls (28%), and miscellaneous (12%)* constitute the major causes of body trauma. The greatest number of cases of abdominal trauma are secondary to auto accidents and direct blows to the abdomen, with only a minor number resulting from falls.

The types of abdominal injury are best considered under two major subdivisions: blunt and penetrating wounds caused by either direct or indirect forces. It is important to consider abdominal injuries in this manner because of the high mortality rate associated with blunt injuries to the abdomen. This is frequently due to the difficulty in diagnosis of these injuries and their lethal potential if not recognized and treated adequately.

In one series of 152 cases of trauma, there was a 6.7% mortality rate with penetrating wounds and 54.7% rate with blunt injuries and in 61% of these deaths, abdominal trauma was the sole cause.

GENERAL PRINCIPLES AND DIAGNOSIS

The diagnosis of serious damage to intra-abdominal structures, is not always possible soon after an accident. Often the intensity of the pain is not proportional to the injury sustained. Symptoms may be masked in the unconscious patient, or may be missed in the patient with other more obvious injuries. Therefore, patients brought to hospital with any history of an abdominal injury should be detained for at least six hours, no matter how slight the trauma may have been. Observations include half-hourly pulse and B.P. recordings with two thorough abdominal examinations before the patient is released. Examination of the urine will usually rule out injury to the kidneys, ureters and bladder.

When signs of abdominal injury are evident and shock has been adequately treated, the following procedures should be considered as aids to diagnosis and treatment.

1. *Gastric Aspiration:* Gastric aspiration is done to look for blood which is presumptive evidence of injury to the stomach or duodenum and to reduce gastric dilation which often occurs early in abdominal trauma and can simulate the signs and symptoms of the acute surgical abdomen. Decompression of the stomach also reduces the anesthetic complications of aspiration, should surgical treatment be required.

2. *X-Rays:* Three views of the abdomen are usually done. The presence of free air suggests viscus perforation. Loss of the psoas shadows may indicate the presence of free peritoneal fluid. Other shadows may imply a localized collection of blood.

It is necessary to rule out radiographically, other injuries which may present with all the symptoms we associate with abdominal trauma. A chest film preferable in the upright position should be done for this purpose. A search for a fractured transverse process or pelvis in other films should be made, as both of these may give all the signs of intra-abdominal injury.

An I.V.P. must be done in all patients when their urine contains blood to rule out trauma to the kidneys, ureters and bladder.

3. *Aspiration of Peritoneal Fluid:*[11] In the absence of profound abdominal distention, an eighteen gauge needle or a plastic catheter may be passed into four lateral quadrants. This is estimated as being 85% accurate in the diagnosis of intra-abdominal hemorrhage by some authorities if positive, but a "negative tap" is meaningless.

4. *Exploratory Laparotomy:* If no improvement in the patient's general condition occurs despite resuscitative measures and a reasonable possibility of an abdominal lesion exists, the advice of Sir Cuthbert Wallace that it is safer to look and see than to wait and see is considered sound.

FREQUENCY OF ORGAN INJURY

According to J. F. Perry,[9] the frequency of organ injury is as shown in Table 1.

Organ	*Blunt* %	*Penetrating* %	*Abdominal Trauma* %
Spleen	30%	5%	35%
Liver	10%	18%	28%
Small Intestine	6%	6%	12%
Bladder ...	6%	1%	7%
Large Intestine	1%	6%	7%
Pancreas ...	1%	1%	2%
Miscellaneous ..			9%

The most significant of these will be discussed in the following paragraphs.

SPLENIC TRAUMA

As can be seen from Table 1 the spleen is the single most frequently damaged organ in cases of abdominal trauma which are admitted to the hospital. The greatest number of these injuries is due to blunt rather than penetrating force.

In a large series of cases reviewed by Whitesell, he observed that 90% of cases of splenic rupture presented within 6 hours of injury and the other 10% within one to six days after injury. Three quarters complained of a constant left upper quadrant pain of increasing intensity. A minority of these had LUQ pain decreasing in intensity at the onset but recurring one to several hours later, rapidly increasing in intensity at that time. Forty per cent of these cases with pain also had left shoulder tip pain which could often be elicited in the Trendelenburg position. Thirty per cent of cases presented in shock on admission. Other accompanying signs and symptoms were those due to blood loss into the peritoneum. i.e., those of the acute surgical abdomen.

The mechanism of injury in most cases of splenic rupture due to blunt trauma is a blow to the left lower chest, left upper quadrant of the abdomen, or fracture of the lower left ribs which causes shearing of the splenic vessels or an extensive capsular tear.

Rupture of the spleen is a very serious injury because of the volume of blood lost and the rapidity with which the patient goes into hypovolemic shock. About 10-15% of the cases of splenic rupture present as a delayed rupture as was mentioned, one to six days after injury, for reasons not completely understood. Two mechanisms have been put forward to explain the delay in the appearance of signs and symptoms of splenic rupture both of which involve the idea that an intra capsular tear can occur. Bleeding continues until it forms a hematoma of sufficient size to rupture the splenic capsules and manifest sudden symptoms. Otherwise a coagulum seals a rent in the capsule temporarily and prevents overt bleeding immediately, again to give a picture of delayed rupture.

Treatment of the ruptured spleen is splenectomy with adequate exploration of the other abdominal organs for associated injuries. It is well to observe at this time that the rigid abdomen may be the only sign in the unconscious patient of such a lethal injury as a ruptured spleen. Until better methods of detecting injury are developed, it is probably wise to follow the premise stated previously, that it is better to explore the abdomen with minimal indications than to overlook lethal injuries of this nature.

LIVER TRAUMA

Referring again to Table 1, it is obvious that liver is the most frequent solid viscus injured by penetrating wounds and so is a significant part of any discussion of abdominal trauma. Inspite of increased interest in the diagnosis and treatment of liver injuries, the mortality remains unacceptably high. Approximately one third of patients with blunt liver injuries die before reaching the hospital, one third die in hospital, and one third survive.

In a review of 303 cases of liver trauma the commonest causes were penetrating wounds from stabs and gunshots (75%), and blunt trauma (25%)[5] from automobile accidents. The right lobe is affected five times more frequently than the left because of its more vulnerable position and larger size.

Because of its rich blood supply, haemorrhagic shock usually dominates the clinical picture. Aspiration of peritoneal fluid from the right upper quadrant may show blood in the peritoneal cavity as may abdominal X-rays (e.g. loss of psoas shadows). The right hemidiaphragm may be elevated or fractures of the right lower ribs may direct our attention to liver injury.

The mechanism of injury is related to sudden displacement of the liver upward into the chest and downward into the abdomen with resultant tearing of the liver at its ligamentous attachments.

The clinical picture usually includes: pain localized to the right upper quadrant then becoming more generalized and often referred to the right shoulder tip, tenderness and rigidity with guarding, percussion dullness, decreased bowel sounds, paralytic ileus and abdominal distention (usually occuring late) with the overall picture of continuing blood loss.

The treatment is immediate surgical repair. At laparotomy, temporary control of hemorrhage may be obtained by having the assistant insert his finger into the Foramen of Winslow and pinching the free edge of the gastrohepatic membrane thereby compressing the portal vein and hepatic arteries. Devitalized tissue is removed and this helps to reduce the post operative complications (see later). The tear in the liver may be sutured with or without the use of absorable gauze (Felfoam) to prevent the sutures from cutting into the liver tissue and to help stop the bleeding. The liver holds sutures poorly and great care must be taken in the repair.

It is considered important that surgery include drainage of the common bile duct to prevent a collection of bile at the site of trauma with later rupture and bile peritonitis. This drain is usually removed on the fifth day.

From a review of 100 cases of liver wounds[10] complications fall into four main categories: infection, hemorrhage, avascular necrosis with disruption of liver tissue, and abnormalities in biliary drainage. The commonest post-operative complication is infection i.e. intrahepatic abscess, subphrenic abscess, peritonitis or wound infection. Hemorrhage may occur from the wound and may present as hemobilia, a rare complication usually heralded by biliary colic three to four weeks after the trauma due to the passage of blood clots down the biliary ducts from a hematoma in liver parenchyma. Secondary hemorrhage may occur from necrosis and infection of hepatic tissue. Bile peritonitis occurs where adequate post-operative drainage is not maintained.

ABDOMINAL TRAUMA

The need for rapid diagnosis and treatment is evident from the large number of patients who die of hemorrhage. However it should be noted that liver trauma is often associated with multiple intra-abdominal injuries. Since most of the patients with liver trauma die of hemorrhage from associated injuries,[5] improved results are most likely to accrue from more rapid diagnosis, vigorous correction of shock and careful control of bleeding at surgery.

SMALL BOWEL TRAUMA

Blunt and penetrating injuries can involve the small bowel. Penetrating injuries can occur from without or within.

Blunt injuries are usually secondary to crushing, tearing (tangential force) or compression.

Tears may occur anywhere around the 360 degree diameter of the bowel but often occur on the posterior surface facing into the mesentery due to its relative fixed position and relationship to the rigid vertebral bodies. Usually the tears from indirect violence occur at the points of bowel fixation, i.e. the upper jejunum near the ligament of Treitz or the distal ileum.

Clinically the picture includes one of the hypovolemic shock secondary to the blood loss or outpouring of intestinal contents which also gives a peritonitis. Pain and tenderness in the hypochondrium and radiating to the respective shoulder tip often occurs. Rigidity of the abdominal musculature with shifting dullness in the flanks and nausea with vomiting may also be present.

On X-ray, the stomach may appear dilated and gas fluid levels may be seen. Haziness of the psoas shadows may also be noted as well as free air under the diaphragm. In some patients an upper GI series (using a small quantity of hypaque) may confirm the diagnosis of a retroperitoneal dudenal tear. These tears can easily be missed in the third part of the duodenum, even at laparotomy. The mortality from such duodenal injury remains high owing to difficulties in diagnosis and to associated injuries, especially to the pancreas. Usually the diagnosis is not readily obvious but it should always be suspected in patients observed to have signs of hypovolemic shock, peritonitis and pancreatitis because if it is diagnosed and treated within six hours, the mortality is only 10%.

Surgical intervention is the treatment of choice. Treatment of shock and nasogastric suction are begun simultaneously. At surgery, hemostasis is necessary in order to make an adequate inspection of the stomach, duodenum, small bowel, colon, and mesentery. Usually the wound edges are trimmed and the bowel closed transversely but resection may sometimes be necessary.

With penetrating wounds of the bowel, assessment is difficult and the abdomen is usually explored surgically on admission. Diagnosis may be aided by x-ray with evidence of pneumoperitoneum, but it is better once again, to explore a negative abdomen than to miss a serious penetration of the bowel.

Occasionally swallowed objects produce perforation but generally they pass right through the intestine. Treatment in these cases is usually expectant with multiple x-rays to follow the object's progress. If the patient develops sudden pain, tenderness, fever or elevated W.B.C. or if the object fails to make progress, surgical exploration is indicated.

STOMACH TRAUMA

Stomach trauma is rare in the absence of other injuries and usually occurs in patients with over distended stomachs (beer drinkers). Often simple suturing is adequate but partial resection may be necessary.

KIDNEY TRAUMA

Renal damage in cases of trauma is usually of secondary importance to injuries of other organs. In fact, in one series of 478 cases of abdominal trauma[2] only one death was directly attributable to renal injury. The low incidence of significant renal injury in abdominal trauma is due in part to the sheltered position that the kidneys occupy guarded by the lower ribs, spine and vertebral muscles and partly due to the protection offered at least in adults by the perirenal fat and Gerotas fascia. The lack of the latter in children plus a greater degree of renal ptosis make their kidneys more susceptible to damage. However, males in the second to fourth decade comprise the greatest number of patients suffering from renal trauma since their activities are strenuous and their kidneys occupy such a fixed position retroperitoneally.

Renal trauma as with other organ trauma is divided into penetrating wounds (the majority being secondary to gunshot or stab wounds which usually damage other organs more seriously) and blunt injuries. The blunt injuries may involve direct forces, as in direct blows suffered in sports and in automobile accidents. Indirect forces such as the vertical decceleration after a fall in which the kidney continues in its downward path in the retroperitoneal space and is torn from its pedicle, also are a cause of blunt trauma.

The signs, symptoms and diagnosis of renal trauma are similar in nature, though not necessarily in severity regardless of the degree of damage done. Damage varies from simple renal contusion to rupture of the renal pelvis. These signs and symptoms consist of one or several of the following: local tenderness, costovertebral angle pain or upper quadrant pain with possible radiation into the groin, muscle spasm in flank and upper quadrants, flank swelling, hematuria and symptoms of shock if hemorrhage is present.

Of the above, hematuria is the most constant finding occurring in 427 cases out of 478 cases of abdominal trauma[2]. The absence of hematuria however cannot be used to exclude renal trauma because the respective ureter of a traumatized kidney can be severed or obstructed with blood clots or peripheral parenchymal damage may not communicate with the collecting system. Assessing the extent of injury to the kidney is then best completed radiographically with plain films of the abdomen (KUB) and an I.V.P. checking for such signs of pathology as an enlarged renal shadow, an obliterated psoas shadow, lateral spinal deviation, a hazy renal outline, failure of dye to be concentrated by the kidney, distorted collecting systems or extravasation of dye.

The types of pathology encountered in cases of abdominal trauma causing renal damage are several. Simple parenchymal contusion is by far the most common, comprising 64-66% of renal injuries in two different series.[2] With contusion, the renal capsule is usually intact with the kidney outline normal with I.V.P. and no x-ray evidence of extravasation. Rupture of the parenchyma inevitably includes tearing of vessels and usually presents as hematuria. With an intact capsule, hemorrhage is subcapsular and will often appear as an enlarged renal shadow on plain film. With capsular tears, a perirenal hematoma can form, giving a hazy renal shadow on x-ray or appear as an obliteration of the psoas shadow. Hemorrhage can be severe but unless the tear extends into the pelvis and calyces, no urine extravasation is present. Rupture of the renal pelvis or calyces usually results in a large extravasation of urine visible on I.V.P. These ruptures are generally due to indirect trauma in which the pelvis of the kidney is jammed against the ribs or vertebral transverse processes. A small extravasation of urine may be absorbed, but the larger ones may develop into perinephric abscesses consequent to invasion by micro-organisms. Patients with tears of the hilus of the kidney (common in penetrating injury involving the renal artery) usually do not survive long enough to reach hospital because of extensive hemorrhage.[2]

In general, then treatment of other associated injuries in cases of abdominal trauma is more important than dealing with the kidney lesion which often heals without interference. Usually renal lesions are treated conservatively, bed rest for a period of two weeks to prevent secondary hemorrhage, and adequate blood replacement and antibiotic therapy.

Exploration is definitely indicated in the presence of one or more of the following signs and symptoms: a falling hematocrit, a renal mass increasing in size, a falling blood pressure with a rising pulse, signs of infection in cases where extravasation is demonstrable, and severe hematuria with or without colic.

Surgical treatment of renal damage usually consists of drainage, partial nephrectomy or nephrectomy for persistent hemorrhage, multiple deep parenchymal lacerations or pedicle damage. This procedure, of course is done after adequate examination by I.V.P. for a contralateral functioning kidney.

PANCREATIC TRAUMA

The pancreas occupies a relatively protected position in the abdomen and therefore is involved in only about 2% of abdominal injuries. The incidence of penetrating trauma slightly exceeds that of blunt trauma to the pancreas.

A confirmed diagnosis of isolated pancreatic trauma in penetrating and blunt injuries is rarely made prior to surgical exploration. The symptomatology of pancreatic trauma is delayed several hours until acute pancreatitis, due to release of enzymes secondary to tissue and duct disruption, develops. An index of suspicion concerning the possibility of pancreatic disruption and continued observance of serum amylase levels will aid in diagnosis of pancreatic trauma.

Treatment of pancreatic injury is usually surgical, the procedure of choice depending on the site and extent of injury. Control of hemorrhage, external drainage with or without duct repair, removal of devitalized tissue, and possible provision for of secretions (or viable separated distal fragments) are all part of the surgical procedure.

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Symptomatology

The Acute Knee

The knee is the most frequently traumatized joint of the body, primarily because of its complex mechanism of action and its vulnerable position. A disproportionate number of knee injuries are seen during the football season, and so a brief discussion of the commoner probelms would be appropriate at this time. This article will deal with cases presenting with a painful knee, following a history of trauma, exclusive of fractures involving the joint.

I STRUCTURE AND FUNCTIONS

Because of the complexity of this joint, a firm knowledge of its anatomy and functions is necessary in interpreting the history, the mechanism of injury, and in arriving at a diagnosis.

The knee is unique in that it combines great stability and great mobility, two features which are usually incompatible in a joint. The arrangement of the bones and ligaments accounts for this.

There are three bony articulations: the patella and the femur, the lateral and medial tibial and femoral condylar articulations. Because of the difference in curvature of the femoral condyles, the knee is regarded as a modified hinge joint, allowing not only flexion and extension, but also some rotation on the tibia, and slight gliding and varus-valgus rocking motions when in the semiflexed position.

The joint capsule is a complicated, incomplete structure including fibres from the true capsule, the tendons of the quadriceps and semimembranous muscles, the fascia lata and iliotibial tract, plus the two collateral ligaments. The medial ligament consists of a superficial group and a deep one, which also attaches to the medial meniscus. The lateral ligament is cordlike and attaches to the head of the fibula, splitting the biceps tendon. These ligaments are very important for stability in extension when they are most tightly stretched and prevent rotation.

Three intra-articular structures must be described: the anterior and posterior cruciate ligaments, the lateral and medial menisci, and the synovium.

The anterior cruciate prevents posterior displacement of the femur on the tibia and the posterior plays a converse role. In flexion, the ligaments are less taut, allowing gliding of the tibia on the femur; this is a position often seen in motorcycle and automobile accidents. An addition to the supportive function of the ligaments is their major contribution to the propioceptive sense, a point which must be remembered when they are repaired with other non-ligamentous structures.

The cartilaginous menisci help compensate for the incongruence of the bony surfaces, and their wedge shape aids in the lubrication of the joint surfaces. The convex border of the medial meniscus is well anchored to the deep surface of the capsule.

The synovial membrane consists of four freely communicating areas, making up a surface of 43 square inches. There are a central, a suprapatellar, and two posterior pouches. Surrounding the joint anteriorly and superiorly is a series of three prapatellar bursae. Since the stimulus for production of synovial fluid is movement of the joint, and since the articular cartilage of the joint is nourished primarily by the fluid, long immobilization may be harmful to the cartilage. This point should be remembered in the treatment of knee injuries.

Because of the difference in the curvature of the femoral condyles, towards the end of extension when the foot is fixed, there is a medial rotation of the femur on the tibia involving the anterior cruciate and the vastus medialis. Thus the knee is screwed-home or 'locked', rotation is prevented, the medial collateral ligament tightens and the medial meniscus is drawn medially. It is obvious that with a sudden violent movement the latter may be pinned and split longitudinally.

When flexing, the joint must first be 'unlocked', an action carried out by the popliteus muscle. There is a range of 140 degrees of voluntary flexion plus more with forcing or squatting. A maximum of 50 degrees of rotation is allowed when the knee is flexed to a right angle.

Of the above structures the most commonly injured and thus the most important clinically are: the medial collateral ligament, the anterior cruciate, the medial meniscus, and the prepatellar bursae. Injuries to these structures will be stressed.

II INJURIES AND DIAGNOSIS

As can be seen by the functions of the bones and ligaments, an accurate history of the mechanism of injury is vital in interpreting the features seen on examination.

A *Synovial Injury*

The more important injuries usually present with a swollen, painful knee with more or less incapacitation. Since a synovial effusion is often present an observation of traumatic synovitis is only a sign. All other injuries must be ruled out to make it a diagnosis. It must be remembered that the presence or not of an effusion and the size or tension of the swelling does not parallel the severity of the injury. Such an isolated injury of the synovium is usually the result of a direct blow and should be differentiated from a traumatic hemarthrosis or a prepatellar bursitis. With traumatic synovitis, the patient will complain of acute pain and of swelling fol-

lowing a delay of several hours after injury. A patellar tap sign may be elicited. With a traumatic hemarthrosis, blood usually remains fluid in the joint and should be aspirated. There is immediate swelling, discomfort rather than pain, a rise in local temperature, and perhaps systemic upset. Prepatellar bursitis or "housemaid's knee" may be seen following long periods of unaccustomed kneeling or a direct blow. There is a local serious effusion, pain, swelling and tenderness.

B *Meniscus Injury*

The medial meniscus is commonly injured in the contact sports such as football, hockey, basketball and skiing. The menisci are more vulnerable when the muscles are fatigued. The mechanism of injury involves external rotation of the leg with the knee flexed and the weight on the foot, a position often assumed in all the above sports. Findings include marked and prompt vastus medialis atrophy, immediate incapacity, local pain especially on weight bearing, a protective limp, immediate swelling, tenderness at the joint margin, possibly a click heard or felt during motion, and a usually negative X-Ray picture. The classical sign of locking of the joint short of full extension may be found only immediately after injury or much later, after initial physical examination. In one study, 59% of the cases had no history of locking at all. The history of the injury is most important here since the integrity of the other ligaments may determine the future of the joint. The lateral meniscus is very much less often injured, although Amako in a 1960 study in Japan found a 9 to 1 ratio of lateral to medial injuries.

The most important tears in terms of immediate disability and future implications are the longitudinal ones. The most common ones however, are found in an older age group averaging around 36 years and are horizontal. These are considered to be of degenerative origin following a traumatic incident. Total removal is indicated once the diagnosis is assured.

CLASSIFICATION OF MENISCAL INJURIES

A. *Condition of cartilage:*
- i) Congenitally abnormal (lateral meniscus mostly)
- ii) Degenerative (lateral meniscus tears are mostly degenerative)
- iii) Regenerative
- iv) Normal (medial meniscus mostly)

B. *Isolated Injury:*
- i) Longitudinal Tear (traumatic, younger age group more important) — Anterior or Middle
  - (a) of the periphery
  - (b) of the body
- ii) Horizontal Tear (degenerative, older age group, more common) — Posterior or Complete
  - (a) sagittal
  - (b) coronal cleavage
- iii) Combined
  - (a) pedunculated anterior horn
  - (b) pedunculated posterior horn

C. *In Association With:*
- i) Collateral Ligament Injury
- ii) Cruciate Ligament Injury
- iii) Fracture of Tibial Plateau

C *Cruciate Injuries*

The anterior cruciate ligament is the most common ligamentous tear but not the most important. It may be torn by a forward thrust of the tibia with the knee flexed and internally rotated. There will be a feeling of weakness and uneasiness on weight bearing, little pain or limp, and forward relaxation of the tibia on the femur with flexion, giving a forwards drawer sign. The degree of this mobility depends on the condition of the medial collateral ligament. A controversial point is the necessity of repairing the anterior cruciate ligament for a stable joint. The evidence of Legedahl in Sweden, O'Donaghue, and of some anatomical studies supports the importance of the ligament in normal joint function. However, it is very difficult to diagnose an isolated tear of the anterior cruciate or one in conjunction with a torn meniscus. Contrast arthrography in skilled hands can be a very useful diagnostic aid.

D *Medial Collateral Injuries*

Sprains of the medial collateral ligament are common and important since they are often misdiagnosed and may go on to serious complications. Again the history is important, with a mechanism of external rotation in slight flexion usually being the story. There is swelling and vague tenderness maximal at the femoral attachment. A complete tear is the most potentially disabling of the common lesions and is rarely isolated, usually being accompanied by cruciate tears, loss of the peripheral attachment of the medial meniscus, or fracture of the lateral condyle. The history is one of severe nauseating pain with a tearing sensation. The patient walks with the limb extended and there is usually a hemarthrosis, unless the effusion has drained into surrounding soft tissue through a torn capsule.

SUMMARY

A consideration of the specific conservative treatment and operative procedures in each injury is not possible in the space at hand. Reference should be made to appropriate orthopedic texts.

In summary, all acute knee injuries, presenting as they do with confusing and overlapping signs and symptoms, the exact nature of the mechanism of injury and a good knowledge of the anatomy of the joint is imperative for adequate treatment and minimizing future disability.

P. G. Hodgetts, 7T1

ACKNOWLEDGEMENT

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Emergency Treatment of Multiple Injuries

C. B. Gates 7T0

Serious injury, with resulting disability and death, is now the nation's most important health problem. In the first half of life, accidental injury is the leading cause of death, with traffic and industry causing the highest number of these injuries. Because of this, it is necessary for medical personnel to be active in both the prevention and the treatment of injuries inflicted by these sources. Injuries are often superimposed on pre-existing illness, alcoholism, or drugs such as steroids, antihypertensive agents and anticoagulants. All these conditions add their complications to the patient's assessment and management.

In a recent survey of deaths following traffic accidents in England, the victims sustained an average of greater than three major injuries. One third of these people died at the time of the accident, and another third died within the first twelve hours after the accident, often solely due to exsanguination, from either obvious or concealed hemorrhage. If there is to be a significant decrease in the mortality, then multiple injuries have to be treated rapidly and vigorously.

PHASES IN CLINICAL STATE

There are two clinical phases of trauma: the phase of acute resuscitation, and the phase of stabilized recovery. In the acute phase, there is a marked interference with the body's vital functions. During this phase, the patient may deteriorate rapidly, and so must be frequently examined. In the more stable phase, the vital signs remain constant. There is a prompt and significant loss of body weight of up to ten per cent or more following massive injury, with increased metabolic expenditure. Thus the patient's nutrition continues to present a problem during the stable phase. Although this must be considered in the long-term management of these people, it will not be discussed here.

PRIORITIES IN TREATMENT

The aim in treating multiple injuries is to adequately assess and restore the patient to normal as quickly as possible. Always it is important to consider the patient as a whole, and in multiple injuries the need is more evident than it often is in other illnesses. In the immediate assessment of these patients, the organ systems involved should be evaluated in an orderly fashion, according to a list of priorities[3]:

1. Airway
2. Bleeding and Shock
3. Consciousness
4. Digestive Organs and Abdomen
5. Excretory Organs
6. Fractures

Each of these systems must be regarded from the time of injury at the scene of the accident, in the hospital emergency department, and, if necessary, in the operating room. While speed is necessary to achieve the goal mentioned above, it is *not* as vital as having a well organized approach to the problem of treating these people!

HOSPITAL FACILITIES

Although the emergency department of a large modern hospital has now expanded to serve patients with minor injuries as well as those who are critically injured, the department must be organized to cope with those who come in seriously injured. The planning of an adequate emergency department should consider the following items:

1. Space to handle the injuries
2. Staff, including a general surgeon in charge of general treatment and decision-making, surgeons available from the various specialized fields, and nursing staff who are trained to care for patients who have received multiple injuries.
3. Communication, both with ambulance services and with other departments in the hospital.
4. Equipment for emergency treatment and assessment of the injuries.
5. Availability of the patient's previous medical records and good reference books for the staff to consult, to help assess complicating factors.

AT THE ACCIDENT SCENE

Successful treatment of patients with multiple trauma begins at the scene of the accident. Ambulance personnel must be instructed to deal skilfully with the most urgent problems of respiration, by clearing of the airway and artificial respiration, and circulation, by the application of pressure to open bleeding. They need to have training in transferring patients carefully, to prevent any additional injury, particularly to the spinal cord. If the patient is now placed on a stretcher, unnecessary moving later is avoided. Open bone wounds should be covered with sterile dressings to minimize infection, and limbs splinted to prevent further tissue damage.

IN THE EMERGENCY DEPARTMENT

When the patient arrives in the emergency department, the resuscitation which was begun at the scene continues. A rapid and more complete examination, looking at the various organ systems in order of their priority, provides an accurate assessment of any injuries and suggests further treatment which might be necessary.

The patient can be intubated, and put on a mechanical ventilator if ventilation is still a problem. Examination of the chest may reveal contusion, mediastinal shift, absent breath sounds, tenderness of rib fractures, dullness of fluid, tympany of a penumothorax, or crepitus of subcutaneous air. In all patients, except those with an acute airway problem, a chest X-ray should be obtained, and then any injury can be treated appropriately. The indications and technique of inserting an intercostal chest tube should be familiar to every doctor.

Damages to the circulatory system should be considered next. A person may lose between ten and twenty per cent of his blood volume and still have a normal systolic blood pressure. In order to present with pallor, perspiration, tachycardia and a lowered systolic pressure, a person must lose between forty and fifty per cent of his blood volume. Fluid replacement is thus very important in someone who has received multiple injuries. Saline or dextran solutions are started while blood is being crossed and typed. A central venous pressure line is essential for monitoring the speed of the fluid replacement. Often an electrocardiogram will be valuable in assessing the presence of tamponade, or preexisting disease.

Head injury is often present in people with multiple injury, and coma seriously complicates proper assessment of all organ systems. A continuing record should be made at frequent intervals, to follow changes in level of consciousness, blood pressure, pulse, size and reactivity of pupils, and any abnormal neurological signs. Marked restlessness, which is often associated with cerebral anoxia, may also be due to severe headache, an overdistended bladder, unsplinted fractures, or extensive soft tissue injuries. Central nervous system damage does not cause shock, abdominal rigidity or paralytic ileus, so other sources for these signs must be found, even in the face of head injury.

Injury to the abdominal viscera is often revealed on examination, by the finding of free fluid or air, a mass, tenderness and guarding, or the absence of bowel sounds. Additional information is gained by tapping each of the four quadrants and examining any fluid obtained.

A urinary catheter provides urine for urinalysis as well as a monitor of hourly fluid output. Inability to pass a catheter along the urethra indicates a discontinuity which must be repaired. Gross bleeding should be investigated by the taking of an intravenous pyelogram, which will indicate damage anywhere along the urinary tract.

Fractures of the limbs, trunk or skull should be assessed clinically and by X-ray. A portable X-ray machine will eliminate unnecessary moving and waiting. In association with fractures of the long bones, there may be vascular or nerve injury. It is important that vascular injuries be detected early to prevent the development of gangrene or Volkmann's ischemic contracture. Nerve deficits must be recognized from the time of injury to assess the patient's prognosis.

THE OPERATING ROOM

Sometimes the rapid initial assessment of the patient reveals the need to take the patient to the operating room, as soon as the airway and circulation are established, instead of keeping him in the emergency department or in the X-ray department. Exploration of the peritoneal cavity often reveals concealed hemorrhage, and at this time it is possible to repair major vessels and organs which have been torn. Craniotomy is indicated with a rapid decrease in the level of consciousness, to evacuate an extradural or subdural hematoma.

OTHER MANAGEMENT

After the immediate treatment has been instituted to stabilize the patient's vital signs, attention can then be paid to any other injuries. Fractures and dislocations are now treated. Skin wounds are inspected, debrided, and grafted if necessary. Tetanus prophylaxis should be given. Topical antibiotics may be applied to wounds, although their value is disputable.

SUMMARY

The management of someone who has received multiple injuries is indeed a difficult challenge. All the organ systems involved must be treated, but they should be treated in the order of their importance to the patient as a whole. To do this, one person, usually a general surgeon, must assume the responsibility for the rapid assessment and decisions for treatment. When a well-organized approach is employed, there is a greater likelihood of reducing the mortality and morbidity associated with multiple injuries.

ACKNOWLEDGEMENT

I would like to thank Dr. R. Stone of the Toronto General Hospital for his comments on this article.

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Clinicopathological Conference

Proteinuria In A Paraplegic

CASE PRESENTATION

Mr. I. M., age 33, suffered a spinal injury in December, 1962 when his car overturned. There was no evidence of periods of unconsciousness. Immediately after the accident, he noted anesthesia and paralysis of his legs and pain in his right shoulder. He was admitted to Toronto General Hospital that afternoon. Physical examination and X-ray revealed a fracture dislocation at the T12 level with complete paraplegia, and fractures of the right scapula and of two ribs on his left side. Hemogram, B.U.N. were within normal limits. Urinalysis was normal. Urine cultures on various occasions grew E. *coli,* yeast, *Ps. pyocyaneus, A. aerogenes, Strep. fecalis* and *Staph. albus.* Total serum proteins were 5.1 gm. with A/G reversal, albumin 2.1, globulin 3. Three blood cultures were negative. The total serum bilirubin was 0.8 mgm %. The patient was transferred to the paraplegic unit where he was kept on bed rest for twelve weeks following his injury, at which time he started on full activity. He was discharged to a Paraplegic Hospital and at the time of discharge was feeling well and had no pain in his back.

Up to November 1963, the patient improved considerably. However, during this period, he developed a sore over the right ischial tuberosity. By December, it had become deep and infected. Cultures of material from the area were eventually shown to grow staphylococcal organisms, *B. proteus* and *Strep. fecalis.* The skin over the right great trochanter broke down and that over the left trochanter became red, inflamed and fluctuant. To establish drainage, the area was incised. The patient became febrile and manifested evidence of chest infection. He was treated with Kantrex and large doses of penicillin. His temperature returned to normal on this therapy, but when it was discontinued, it rose once more. He was then placed on Tetracycline. X-rays revealed osteomyletis of the left great trochanter. The fever continued and he lost, in all, about 25 lbs., from 126 down to 101, before being transferred to Toronto General Hospital, acutely ill with septicemia. There, the infection was brought under control by systemic antibiotics and ultimately a rotation flap was carried out, first on one hip and then on the other. The initial sores over the great trochanters responded well to this therapy and remained closed. The patient became afebrile and gained weight (134 lbs.) and was finally mobilized and discharged back to the Paraplegic Hospital in April 1964.

In January 1965, he was again admitted for a pressure sore. This had developed into a 2″ x 3″ ulcer over the left buttock, directly over the ischial tuberosity. Physical examination was essentially otherwise non-contributory. At this time, he had a B.U.N. of 13 mgm %; normal electrolytes and serum proteins; Hb 14.8 gm%; W.B.C. 9,500. X-rays showed no involvement of ischial tuberosities under the infected ulcer. A large rotation flap that healed well was performed. In March, he was discharged to the Paraplegic Hospital.

The patient then managed well until 1967. He began to notice that his appetite was poor and he felt much fatigued and that there was swelling in both legs below the knees. In March, he was again admitted to hospital for decubitus ulcer. At this time, he appeared pale and chronically ill. No abnormalities of chest, heart or abdomen were evident. There was pitting edema from ankles to groin bilaterally. On the left buttock in the region of the previous rotation, there was a large 4″ necrotic sore. This failed to respond to conservative treatment and so another rotation flap was carried out. Post-operatively, he did well and when permitted up, he suffered no ill effects. However, he continued to manifest a low-grade fever. Chest films were normal. B.U.N. at various times: 16, 31, 19 mgm %; serum proteins, total 3.8 gm%, A/G 0.9/2.9; urine protein excretion was 6.39 gm in 24 hours; urine cultured *A. aerogenes* and *B. proteus.* Congo red test was positive for amyloid with 70% of the dye disappearing in one hour, and none appearing in his urine. Hb 81%, 12.3 gm %; Hct 45%; W.B.C. 9,800. When he became afebrile after treatment with gentamycin, he was discharged (June).

On August 15, 1967, he fell while moving from his bed to a chair, broke his left hip, and was again admitted to hospital. He was very thin and pale. His chest and cardiovascular system were normal. He had massive decubitus ulceration of the sacrum and left greater trochanteric area. B.U.N. 67 mgm %; creatinine 2.4 mg %; Hb 6.5 gm %; protein 3.4. Transfusion was used to replace his depleted blood volume and hemoglobin; however, his condition continued to deteriorate. Subsequent investigation: blood sugar 106 mg %; B.U.N. 105 mgm %; total protein 4.2; urine culture positive for Klebsiella, Proteus, Pseudomonas and others. On August 24, the patient went into complete kidney shut-down and from then to August 28, when he died, he put out no urine.

DIFFERENTIAL DIAGNOSIS

In summary, the patient presented with two sets of problems. The first is composed of paraplegia, decubitus ulceration and recurrent infection. The second is made up of (1) profuse proteinuria, (2) extensive edema and (3) hypoproteinemia and hypoalbuminemia. These three features are the components of the nephrotic syndrome.

The nephrotic syndrome is not regarded as a disease in itself. It has multiple causes and may occur as a complication of well-defined pathologic disorders where there is diffuse injury to the glomeruli. In the majority of adult cases, the etiology is unknown. It also does occur with post-streptococcal chronic glomerulonephritis, systemic lupus erythematosus, diabetic intercapillary glomerulosclerosis (Kimmelstiel-Wilson disease) and renal amyloidosis. On occasion, it is observed in reactions to various allergens and drugs. Nephrotoxins such as organic mercurial diuretics, inorganic mercury, bismuth and gold have been implicated. Several infectious diseases show a higher influence of the nephrotic syndrome. These include cytomegalic inclusion disease, syphilis, malaria, typhus, chronic jejunoileitis, tuberculosis, subacute bacterial endocarditis and herpes zoster. Thrombosis of the renal veins or constrictive pericarditis can also produce a somewhat similar picture.

In this case, there was no evidence to implicate any drug or allergens nor was there any history of exposure to a nephrotoxic agent. Although the patient had several episodes of infection during his course, there was no evidence of streptococcal infection nor, indeed, of any of the associated infectious diseases. Few of the expected manifestations of lupus were present. Throughout, no aspect of the physical or chemical picture suggests diabetes or pericarditis. With gradual occlusion of the renal veins, partial function may be preserved and, in a few such cases, there will be massive proteinuria and the nephrotic syndrome. Such occlusion is usually due to (a) invasion of the veins by hypernephroma, (b) compression by malignant metastases to retroperitoneal lymph nodes at the level of the celiac axis, or (c) thrombosis secondary to thrombophlebitis of the legs. Although, there were no signs of sudden complete thrombosis, the presentation in this patient is not incompatible with gradual partial occlusion such as might be due to invasion or thrombosis. The presence of paraplegia in this patient is, of course, a predisposing factor for thrombophlebitis. Amyloid deposition seems to be fairly well established in this patient. Both his physical picture of hepatosplenomagaly and the chemical picture of a positive Congo red test point in this direction. Such deposits in the kidney could account for the symptoms of the nephrotic syndrome in two ways: directly, by glomerular injury and/or indirectly, by predisposing the renal veins to thrombosis.

In conclusion, on the basis of the physical and chemical evidence presented, and weighing the probabilities involved, the following diagnosis is offered: chronic renal insufficiency with failure, due to secondary renal amyloidosis.

AUTOPSY FINDINGS

Gross Examination

Pleural cavities showed adhesions over both upper lobes. The right lung was congested and the left inflated. The peritoneal cavity was found to contain 150 millilitres of straw-coloured fluid. Many foci of fat necrosis were noted on the mesenteric, retroperitoneal and omental fat. The liver was markedly enlarged (2525 grams) with a firm parenchyma and regular surface. The parenchyma of the pancreas was swollen and showed foci of necrosis and hemorrhage. Many areas of fat necrosis were also evident. The spleen was moderately enlarged (480 grams) with a very firm, dark red parenchyma. The left kidney weighed 105 grams and was atrophic. The cortex was thin and there was coarse regular scarring on the surface. The right kidney weighed 300 grams and was markedly hypertrophied with a very firm parenchyma. In both kidneys, the calyces and pelvis were slightly dilated, and contained greenish pus. The bladder was very small, its wall severely thickened by fibrous tissue. In the spinal cord, the dura was thickened and fibrotic at the body of T12. Above and below the lesion, the cord was grossly atrophic, especially at the mid-thoracic level. There was atrophy and fibrosis of both the ventral and dorsal spinal roots.

Microscopic Examination

The lungs showed congestion. Foci of cytosteatonecrosis were seen in the peripancreatic fat. Diffuse amyloidosis was present. This was observed in the parathyroids, thyroid, heart, adrenals, bladder, testes, large bowel, and especially in the liver, spleen and kidneys. Also in the kidneys were sign of chronic pyelonephritis. The wall of the urinary bladder showed fiborsis and the presence of chronic inflammatory cells. Atrophy and fibrosis of the testes were evident. The spinal cord displayed ascending demyelination and gliosis of the fasciculus gracilis and the spinocerebellar tract bilaterally.

Pathological Diagnosis

1—Acute pancreatitis with necrosis of mesenteric and retroperitoneal fat

2—Secondary amyloidosis with hepatosplenomegaly and renal involvement

3—Paraplegia — fracture of T12 with neurogenic bladder, chronic bilateral pyelonephritis and decubitus ulceration.

DISCUSSION

The disorder now known as amyloidosis has been recognized since the early nineteenth century. Until re-

cently, no means were available to diagnose amyloidosis during the life of the patient, and today, our knowledge about this unique syndrome remains quite meagre. Nonetheless, its importance is becoming more evident, and, in a quite extensive recent review (1), it is claimed that it may come to be regarded as one of the major causes of long-term hospitalization. Although its value is now being reappraised, the popular classification recognizes two major categories of disease: primary amyloidosis, a disorder with no antecedent or coexisting disease, and secondary amyloidosis, associated with chronic disease. Several heredofamilial amyloidoses have been described[1].

Secondary amyloidosis has been associated with chronic infectious (tuberculosis, leprosy, syphilis, osteomyelitis, bronchiectasis) and probably infectious (Reiter's Syndrome, Whipple's) diseases, with neoplasms (Hodgkin's disease, multiple myeloma, renal-cell carcinoma, medullary carcinoma of the thyroid) with some metabolic diseases (diabetes mellitus), and especially with chronic inflammatory diseases (rheumatoid arthritis and variants, other connective-tissue diseases, regional enteritis, ulcerative colitis). The clinical manifestations depend on the major site of amyloid deposition.

For many years the association between amyloid and paraplegia has been observed, but only now is this being fully appreciated. One study (2) revealed that 40% of 65 paraplegic patients studied had amyloidosis. Of those with amyloidosis, 70% of the deaths were due to renal failure. On the basis of their own cases and from a review of the literature, they felt that renal failure appeared to be the leading cause of death in paraplegia and furthermore, that amyloid is commonly the agent responsible for the failure. Renal amyloid aggravates the chronic pyelonephritis commonly present in such patients, thereby shortening their lives. Briggs[3] looked at a series of 53 cases of amyloidosis and found paraplegia to be the associated disorder in 45%.

Dalton et al. [2] also felt the underlying lesion in their series to be decubitus ulceration rather than pyelonephritis. Malament et al.[4] suggest that either chronic osteomyelitis as a complication of decubitus ulcers and/or a protein metabolism defect resulting from excessive loss of serum proteins from draining wounds and from urinary excretion may be the causative factor(s) in the development of the amyloidosis. Nonetheless, although a large number of patients with cord injury develop the conditions of decubitus ulceration, osteomyelitis, and pyelonephritis, only those with recurring complications tend to develop secondary amyloidosis. Accordingly, although there is no relation between the level of spinal cord injury or the age of the patient and the incidence of amyloidosis, the duration of complications of paraplegia is an important component.

The treatment of amyloidosis is complicated by the difficulty of diagnosis. There are no laboratory abnormalities specific to or unique for amyloid. There is no single finding in the blood, urine, EKG, or X-ray film specific for this disease. The most popular test has been a serum dye-extraction test using the dye Congo red, but even here, results must be interpreted on an empirical basis. It is now felt that biopsy and the use of appropriate stains is the proper way to establish the diagnosis, although to date this has not been completely successful.

No specific treatment exists for any variety of amyloidosis. Generally, one attempts to cure the underlying disease, although even this does not guarantee freedom from amyloid. Steroids have been found to have little effect on the disease. Prevention of secondary amyloidosis in the patient with spinal cord injury should be directed toward avoidance of formation of decubitus ulcers and toward intensive care of ulcers when present. Chemotherapy and antibiotics are indicated, to be followed by surgical repair to cover the extensive skin defects.

The prognosis of amyloidosis has varied from one to four years in most large series, although a number of five-year survivals have been recorded. Nonetheless, the tools to prevent secondary infection are at hand so that the situation is not hopeless. Every effort should be made to maintain these patients.

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Grand Round Quiz

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Multiple Trauma

1. A patient arrives in the Emergency Department following a motor vehicle accident, in shock. Until disproven, the etiology of the shock would be considered to be:

   (a) Cardiogenic
   (b) Vasovagal
   (c) Hypovolemic
   (d) Neurogenic
   (e) Septic

2. On arrival in the Emergency Department following an auto accident, a patient is found to have sustained the following injuries:

   Fractured skull
   Soft tissue injury to chest wall
   Fractured right side of pelvis
   Blunt abdominal trauma

   The most important aspect of initial management is:

   (a) Immediate intravenous fluid administration
   (b) X-ray studies — i.e. 3 views of abdomen, chest x-ray, etc.
   (c) Adequate and full general physical and neurological examination
   (d) Establishment of an adequate airway
   (e) Control of bleeding

3. In the resuscitation of an 80-year-old patient in shock because of blood loss into a retroperitoneal space from a pelvic fracture, your initial intravenous buid replacement would be:

   (a) Dextran (high molecular weight)
   (b) Dextran (low molecular weight)
   (c) Normal saline (0.9%)
   (d) 5% Dextrose in water
   (e) 3.3 Dextrose and 0.3% Sodium Chloride injection (2/3 - 1/3)

4. Adequate volume replacement, as indicated by a Central Venous Pressure of 12 to 15 cm. of water, in a patient with multiple trauma, has been achieved. However, urine output is still inadequate, the pulse is at 100 per minute, B.P. 90/60 and peripheral vasoconstriction persists.

   You might consider the use of:

   (a) Dibenzyline (Phenoxybenzamine)
   (b) Levophed (Noradrenaline)
   (c) Dextran (40)
   (d) Lasix (Furosemide)
   (e) Isuprel (Isoproterenol)

5. A patient with, among other injuries, multiple rib fractures on the right side, develops rapidly progressive subcutaneous emphysema in his neck.

   Would you:

   (a) Order a bronchoscopic examination
   (b) Insert an underwater seal drain in the right pleural space
   (c) Insert needles into the skin of the neck for decompression
   (d) Order a bronchogram
   (e) Order an esophagogram

6. Of the solid viscera injured in blunt abdominal trauma, the most common is the:

   (a) Liver
   (b) Kidney
   (c) Spleen
   (d) Pancreas
   (e) Testis

7. A patient returns 3 weeks after blunt abdominal trauma complaining of colicky epigastric pain, coffee ground vomitus and dark urine.

   The most likely diagnosis is:

   (a) Mallory-Weiss syndrome
   (b) Hemobilia
   (c) Stress ulcer
   (d) Duodenal hematoma
   (e) Pseudocyst of pancreas

8. A patient who was involved in an auto accident and is known to have a compression fracture of lumbar vertebral bodies 1 and 2, becomes hypotensive and vomits coffee ground material some 12 hours after injury.

   The most likely diagnosis is:

   (a) Retroperitoneal dudenal tear
   (b) Lacerated esophagus
   (c) Bleeding stress ulcer
   (d) Bleeding duodenal ulcer
   (e) Acute gastric dilatation

9. A patient is seen, who among other injuries, has a fractured pelvis. Attempts at catheterization fail. The most likely diagnosis is:
   (a) Ruptured membranous urethra
   (b) Ruptured prostatic urethra
   (c) Ruptured bladder
   (d) Benign prostatic hypertrophy
   (e) Ureteral stricture

10. Your management of a case with a penetrating abdominal wound would be:
   (a) Careful monitoring of vital signs, taking appropriate action with change in them
   (b) Local exploration of the wound
   (c) Urgent exploratory laparotomy
   (d) X-ray studies
   (e) Knife sinogram

11. A patient arrives in the Emergency Department, comatose, in shock, with evidence of a depressed skull fracture, and a crush injury of the left chest wall. His chest x-ray, however, is relatively normal. Initial resuscitation is not very successful.

   You would immediately proceed to:

   (a) Prepare for laparotomy
   (b) Do a rapid neurological examination, x-rays of skull, emergency craniotomy
   (c) Administer I.V. fluids and re-assess patient in 4 hours time
   (d) X-ray studies of abdomen
   (e) Order electroencephalogram

12. In a patient with multiple trauma, the best index of the "shock" state is the:

   (a) Blood pressure
   (b) Central venous pressure
   (c) Clinical evidence of poor peripheral perfusion (cold, clammy, pale skin, etc.)
   (d) Pulse rate
   (e) Urinary output

13. An unconscious patient, with a flail chest on the right, requires intermittent positive pressure assisted respiration. The blood pressure falls, the CVP and pulse rate rise.

   The most likely explanation is:

   (a) Cardiac Tamponade
   (b) Cardiac failure
   (c) This is the usual course of events in a patient on assisted ventilation
   (d) Tension pneumothorax
   (e) Intrathoracic bleeding

14. The sole occupant of a car is brought into the Emergency Department after an accident, smelling strongly of alcohol, restless, confused, with evidence of an anterior chest steering-wheel type injury. On physical examination nothing of note is detected.

   A chest x-ray reveals slight widening of the mediastinum. The most likely diagnosis is:

   (a) Mediastinal hematoma, unrelated to major vessels
   (b) Complete transection of the aorta below the origin of the left subclavian with subadventitial dissection
   (c) Ruptured bronchus with mediastinal emphysema
   (d) Pericardial tamponade after cardiac injury
   (e) Previous disease giving rise to enlargement of mediastinal nodes

(Answers on page 50)

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Answers To Grand Round Quiz

1 — C

This applies to anyone of any age who is suffering from multiple trauma, and it is therefore mandatory to start giving intravenous fluids rapidly, through a well placed adequate intravenous line as soon as possible. Transfusion should be continued at a rate of 1,000 ml. per 5-10-15 minutes until normal volume is restored, as evidenced by a rising CVP, drop in pulse and rising blood pressure.

2 — D

The priorities in the management of a case of multiple trauma are easily remembered as:

A — Airway
B — Bleeding
C — Consciousness
D — Digestive organs
E — Excretory organs
F — Fractures

In thinking about the problem, 3 minutes of anoxia is lethal in a healthy person. Less time is required in the presence of other injuries, especially shock. Bleeding is practically never that massive — but is next in importance.

If the respiratory and circulatory systems are working satisfactorily, all other systems will be able to function unless they are directly involved in the injury.

3 — C

Loss of isotonic fluid, such as blood, must be replaced with an isotonic solution. Although 2/3 - 1/3 and 5% Dextrose in water are initially isotonic and may be attractive because of the undoubted possibility of cardiac disease in an 80-year-old patient, the glucose will be metabolized by the liver. The osmalality of the extracellular fluid, and then the intracellular fluid, will fall. This will impair function of especially cardiac muscle, and it is therefore mandatory that a solution such as normal saline is given.

The use of Dextran is associated with the possibility of decreasing blood coagulation and difficulty in cross matching and is no longer favoured in most centres.

4 — E

Isuprel is a beta adrenergic stimulator, having a positive inotropic action on the heart (increasing the rate and force of contraction and therefore cardiac output) as well as causing a reduction of total peripheral arterial resistance. It should not be used until volume restoration has been completed. Levophed, although it will raise the blood pressure, will aggravate peripheral vasoconstriction and further decrease renal blood flow.

5 — A

Rapidly developing emphysema in the neck should immediately raise the possibility of a ruptured bronchus giving mediastinal emphysema and tracking up into the neck. If the subcutaneous air originated from the rib fractures, one would expect it to start in the chest wall. Bronchoscopy is an easy, fast maneouvre in this circumstance. A bronchogram would be a far lengthier procedure and is contraindicated where respiratory reserve is already compromised.

Esophageal tears can cause mediastinal and hence cervical emphysema, but here there is likely to be a minimal quantity.

A thoracotomy with direct repair of the bronchus is usually indicated with a tear. Bleeding is often associated, as is a tension pneumothorax.

6 —B

Although injury to spleen is the most common cause for exploratory laparotomy following blunt abdominal trauma, kidney injury is manifested by hematuria and abnormal IVP occurs far more frequently. However, in the vast majority of instances, its retroperitoneal position, strong capsule, etc., ensures that bleeding is seldom massive. In fact, even with some leakage of urine from the kidney on IVP, conservative therapy is usually advocated.

7 — B

Lacerations of liver parenchyma occur with blunt abdominal trauma, are often asymptomatic, but at times act in a fashion similar to that of a subdural hematoma in that they enlarge and eventually drain out through a bile duct.

The passage of clots causing colicky pain and transient jaundice with dark urine, terminating in the realease quantities of blood to give hematemesis, and melena is characteristic.

8 — E

An ileus is a very common accompanying event in any condition giving rise to a retroperitoneal hematoma, as would occur in vertebral fractures, pelvic fractures and renal injuries. In almost all cases of multiple trauma, a duodenal tube should be passed, not only to prevent the

complication of acute gastric dilatation, but also to empty the stomach and to prevent aspiration when anaesthesia for surgery becomes necessary. It is also useful as a diagnostic measure, as fresh bleeding may be a helpful clue to unsuspected duodenal or gastric trauma.

9 — A

The urethra most commonly ruptures, as do all hollow muscular organs, at the junction between the mobile prostatic urethra and the membranous urethra. Failure to pass a catheter in this situation calls for urological consultation, a urethrogram and surgical repair. All abnormalities in a patient with multiple trauma must be considered to be due to the traumatic episode until disproven.

10 — C

Although much has been written about the more conservative approaches to abdominal stab wounds, it is still the safest to do an exploratory laparotomy. Most of the other techniques have been developed in centres where many penetrating abdominal wounds are seen daily, and where routine exploratory laparotomy would therefore be impractical.

The methods of careful observation, and sinograms give good results where adequate trained personnel exist. In centres where there is only the occasional stab wound, an exploratory laparotomy is best.

11 — C

Significant shock is never caused by head injury alone unless associated with multiple deep scalp lacerations. Trauma to the left chest wall should in the presence of shock, mean a ruptured spleen requiring emergency laparotomy. A ruptured spleen is the commonest cause for emergency laparotomy following blunt abdominal trauma.

12 — E

All parameters mentioned are useful and should be monitored every 15 minutes and accurately recorded in every case of shock. However, the current concept of shock is that of inadequate perfusion of vital organs and centers.

The kidney is the first 'vital organ' to be 'shut down' in shock and the minute urinary output is a very good index of renal blood flow (½ - 1 c.c. or more per minute). A urine output of 20 - 25 c.c. per hour, which does not respond promptly to an infusion of saline, probably indicates significant hypovolemia. If under 17 c.c. per hour, this is prime evidence of severe hypovolemia and the need for rapid resuscitation.

A cold, clammy skin can occur from neurogenic stimuli not related to central tissue perfusion.
Urine output is, of course, not a good indicator in cases where the injury involves the renal apparatus, or where glycosuria or significant prior renal disease exists.

13 — D

A tension pneumothorax is very likely to occur with assisted ventilation, especially if there has been massive chest wall trauma. The rising intrathoracic pressure causes a rise in CVP and decreased venous return with consequent fall in blood pressure and a rapid pulse. Some authorities advocate the placement of a chest drain in all cases with fractured ribs who are to receive assisted positive pressure ventilation.

A pericardial tamponade, although another possible cause of a similar picture, is far less common. Intrathoracic bleeding would be associated with a fall in CVP initially, although in all patients, assisted ventilation results in some increase of central venous pressure as well as a mild drop in blood pressure. The pulse, however, decreases or stays stable without bleeding.

14 — B

Complete or partial transsection of the thoracic aorta usually at the junction between a fixed and mobile area, e.g. just below the left subclavan take-off, is a relatively common injury associated with sudden stoppage of forward motion. Although there is a complete tear through all layers of the aorta, the adventitial tissue is strong enough to prevent significant leak occurring, so that the patient may appear to be perfectly normal. This points out the need for a chest x-ray in all cases of multiple trauma, especially those where there is evidence of chest trauma. A widened mediastinum in these cases should be taken to be a leak from a partially or completely transsected aorta in most instances and this should be ruled out by means of a retrograde aortogram.

Salvage of these cases is possible by replacement of part of the aorta with Teflon graft. Occasionally these patients are seen months after the injury with large false aneurysms.

For further reading:

1. Early Management of the Critically Injured — D. J. Currie. Canadian Medical Association Journal, Vol. 95: Oct. 22, 1966.
2. Surgical Clinics of North America, Vol. 48 #6: Dec., 1968.

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